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16. ABSTRACT

It is necessary to assure that monitoring measurements are directly related to the population of highest risk and that the major sources of pollutants are clearly identified and quantified. An integrated monitoring system is a systems approach for providing the information necessary to permit efficient control of those sources of pollutants causing major threats to the population of highest risk. A "Workshop for the Design of a Pollutant-Oriented Integrated Monitoring System" convened by EPA in March 1974 summarized the elements of such a systems approach and discussed those information needs yet to be satisfied by basic monitoring research.

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DESIGN OF POLLUTANT-ORIENTED INTEGRATED MONITORING SYSTEMS
A Test Case: Environmental Lead

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I. INTRODUCTION

Environmental monitoring assesses the types and amounts of pollutants in the environment and determines if sensitive human populations or other susceptible receptors are exposed to levels and durations of exposure to pollutants that may cause adverse effects.

It is convenient to define the concepts of "critical receptors" and "critical sources" when discussing the biological effects of pollutants. A critical receptor will be defined to be that subgroup of animals or plants which shows a measurable or observable effect at a dose or exposure of the specified pollutant lower than any other group. A critical source is that single source that contributes more than any other source to the fate of the critical receptor. Typically, but not necessarily, if a critical source is eliminated the debilitating effect on the critical receptor will be eliminated.

It is absolutely necessary to assure that monitoring measurements are directly related to the critical receptors at risk and to critical sources amenable to some control or modification. As we gain more experience and knowledge about pollutants, critical receptors, and critical sources, we also need to carefully review existing, ongoing, and planned monitoring networks to see that they provide the most pertinent data possible.

The concept of an integrated monitoring system was developed to coordinate complex information about the relationships of pollutants, critical sources, and critical receptors. The concept considers many aspects of the problem at once and it defines, quantifies, and compares specific factors about each pollutant.

An integrated monitoring system is a systems approach for providing the information required to permit efficient control of critical sources of pollutants causing major problems or threats in critical receptors. Traditionally most monitoring programs assess only single pathway systems but, in contrast, integrated monitoring attempts to assess total exposure to a pollutant or combination of pollutants.

Design of an integrated monitoring system that will provide the desired exposure assessment requires much specific information. The major areas of concern and the sequence of steps in designing a system include identifying the sources, critical receptors and major transport pathways, determining dosages via major portals of entry into receptors, and identifying and locating major critical sources, especially those amenable to control. In order to organize this information to evaluate the relative importance of different components, a mathematical model appears to be of value. Models will help determine basic research needs and research monitoring requirements, and will help in the design of an integrated monitoring system or network.

It was desired to test the validity of the concept of integrated monitoring systems and to examine the criteria and steps involved. A test case was undertaken by a "Workshop for the Design of a Pollutant-Oriented Integrated

Monitoring System" conducted at the National Environmental Research Center* at Las Vegas, Nevada, in March 1974. Medical and environmental specialists were convened to evaluate the concepts using a selected test-case pollutant.

The pollutant was selected using the following criteria: a) a large amount of data is available on the pollutant and its effects, b) it would probably illuminate most of the problems in designing an integrated monitoring system, c) it should reach man through several environmental pathways, d) the pollutant should cause significant effects in humans, and e) design of a system might be of value for monitoring the selected pollutant. Lead, which seemed to best fit these criteria, was the pollutant selected.

The following report critically examines the concept using this system approach and points out the values and problems. The review pointed out important knowledge gaps, basic research needs, and research monitoring requirements. The use of mathematical models appears to be valuable, but more detailed testing and evaluation of models is required to establish their validity and usefulness.

II. CONCEPT OF AN INTEGRATED MONITORING SYSTEM

The concept of an integrated monitoring systems approach which takes into account simultaneously the critical receptor, critical transport pathways, and critical sources is relatively new. The term "critical" is used to designate a specific and important population at risk and the special transport pathways from the important sources which impact this receptor population. While all sources and pathways are included, instead of general ambient environment monitoring, special monitoring of the major pollutant chain involved is done specifically to permit control of critical sources. This concept includes the following considerations: a) critical receptors, b) critical transport pathways and portals of entry, c) critical sources, d) mathematical models, e) basic research requirements, f) research monitoring requirements, g) design of an integrated monitoring system.

The concept is presented below with a detailed outline of the factors involved and a discussion of models. This outline provided the workshop with the structure for analysis of the concept using the test-case pollutant, lead.

A. Critical Receptor Population

1. Determine Critical Receptor

a. Population with greatest exposure risk (number of persons exposed to greatest concentration of pollutant).

b. Population most sensitive with lowest threshold of effects.

c. Population actually showing most effects.

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* This Center became the Environmental Monitoring and Support Laboratory-Las Vegas effective June 29, 1975.

2. Medical Effects

   a. Determine daily dosage, absorbed dosage, retention, and excretion rates.

   b. Determine minimum threshold of effects of pollutants for both acute high dosage and chronic low dosage in adults and children.

   c. Determine changes and trends in body burden levels and effects with time.

B. Critical Transport Pathways and Important Portals of Entry into Receptor

   1. Determine dispersion and dissemination of pollutant into the environment.

   2. Determine major transport pathways and special areas of accumulation or concentration of pollutant in the environment.

   3. Determine critical pathways from critical sources to critical receptor.

   4. Quantitate total amounts and rates of flow of pollutant in critical transport pathways in different media.

   5. Quantitate special pathways and portals of entry, e.g. placental transfer from mother.

   6. Quantitate amount of pollutant entering (the portals of entry) of the receptor.

C. Critical Sources

   1. Identify sources, both natural and man made, and develop an emissions inventory.

   2. Identify critical sources which are of importance to the critical receptor via identified major transport pathways.

   3. Determine mass emission rate for each major type of source, both mobile and stationary.

   4. Determine chemical composition and particle or aerosol size distribution for each major type of source.

   5. Determine changes of sources and trends with time.

D. Quantitative Mathematical Models

1. General Model

A mathematical model can be of great value in helping to select from various alternatives the relative importance of various sources, pathways, and routes of entry of a pollutant into a receptor. It may be of value in showing the use and limitations of presently available quantitative data and showing the relative importance of obtaining required missing data. The model can help in assigning priority in determining research data requirements, research monitoring requirements, and in design of an integrated monitoring system.

A deterministic mathematical model appears to be suitable for simulating pollutant dispersal to a receptor. This type model usually requires much fewer data than statistical models. However, follow-up experiments for validating the model are required in order to assure the adequacy of the deterministic model. A hierarchical systems approach is used in which the top echelon general model covers the entire system. Lower levels of the hierarchy of models include submodels for specific components which interact with and become an input to the general model.

2. Submodels

Submodels can be developed for each process or compartment of the generalized model. Of particular interest in the integrated monitoring system is the transport or distribution model from the source through the environment to the receptor.

A submodel for transport and diffusion can be developed which includes only diffusion from one or more sources. Submodels can also include entry and absorption in the receptor. A number of atmospheric transport models have been developed. Mathematical diffusion models for specific metropolitan areas have been developed with varying degrees of sophistication. Examples are the Travelers Research Corporation model (Hilst, 1969) and the Argonne National Laboratory model (Roberts, et al., 1969) which are short-term period models with daily predictions of concentrations. An air quality diffusion model by Martin and Tikvart (1968) permits calculation of seasonal or monthly concentrations of any pollutant.

A submodel on atmospheric pollutant transport was developed by Battelle (Lutz, et al., 1970). This represents steady state airborne transport from several sources. Equations for meteorological transport of a pollutant were developed for a) continuous point source, b) finite crosswind line source, c) finite downwind line source, and d) finite aerial source. The validity of the use of models for lead distribution and transport is discussed in the lead case study. In addition, a model was developed at the workshop specifically for lead, and it is also presented.

3. Use of Models

More effort on developing and testing of models and submodel components is required to fully utilize the value of this approach.

During this workshop, the submodel for atmospheric lead concentration was developed but not tested. It provides a theoretical basis for development of an atmospheric monitoring system for a pollutant. In the lead test case, many quantitative data were compiled relating to the sources, critical receptors, and pathways and portals of entry. However, it was not possible to apply the data to any of the models to test them. A complete set of hierarchical models for the special receptor and the special pathways is needed, which would contribute greatly to development of an integrated monitoring system for lead.

E. Basic Research Requirements

Basic research, as used here, is the scientific study of a specific problem to obtain knowledge to understand the function, action, or part played by a component of a system. Basic research may be closely related to research monitoring because it may involve repetitive measurement over a short period of time.

Basic research, for example, might be carried out on the pica behavior (the craving for and eating of unnatural substances) of a child to determine the amount of house or street lead-containing dust the child consumes or breathes. This may be conducted jointly with research monitoring on the daily levels of lead in dust in the houses and on the streets involved in the research program. Both programs are limited in space and time until specific questions have been answered.

F. Research Monitoring Requirements

Research monitoring is the repetitive sampling of a pollutant, or of the effects it produces, to answer specific questions in a research or monitoring program. Research monitoring is usually limited in both time and space, in contrast with routine monitoring of the ambient environment which uses large numbers of sampling stations in a monitoring network.

Research monitoring may be established to answer specific questions on determining the importance of a specific transport pathway or concentration area or, for example, determining the proper height of a sampler to quantify the impact of air particles on the child.

G. Design of Integrated Monitoring System or Network

After identification of the critical receptors, pathways, sources, and critical sources, and after compiling and evaluating all available information (preferably with the use of models), the principles of monitoring systems design may be applied in order to design an integrated monitoring system or network. Various basic research and research monitoring needs would have to

be identified and satisfied before a fully effective and efficient system could be implemented. By following this procedure, one could feel reasonably certain that the monitoring system would yield the data for which it was designed, and that such data would be sufficient for effectively controlling critical sources.

III. EVALUATION OF CONCEPT USING LEAD AS A TEST CASE

To evaluate the concept of an integrated monitoring system, the available data on lead were compiled following the outline presented in Section II. Mathematical models for lead were examined. The applicability of the concept to the specific test case was reviewed.

Lead is a heavy metal which is highly toxic to human beings when absorbed. It is the only heavy metal that does not have some modern therapeutic application and is not essential to the nutrition of human beings, other animals, or plants. The modern interest in the physiological properties of lead centers around its toxicological properties.

Lead is extremely useful and is used in very large quantities. In 1968 the United States used 1.39 million tons of newly produced lead plus 0.55 million tons from secondary sources. (Lutz, et al., 1970)

CRITICAL RECEPTOR POPULATION

Large numbers of people are exposed to lead, but it must enter the body and be absorbed to be dangerous. People who work with lead in mines, smelters, factories, and processing plants are occupationally exposed and may exhibit toxic symptoms. In addition, workers with tetraethyl lead gasoline additive, storage battery workers, ship scrapers, painters, consumers of lead-contaminated "moonshine" whiskey, and other exposed persons such as tunnel workers and garage men, sometimes suffer from lead poisoning. Since there are now quite strict environmental health controls on lead exposure in industry, the number of cases of lead poisoning is not as great as previously. However, children are also known to be subject to lead poisoning, apparently without any more exposure than urban adults.

During the past few years, environmental lead has been the subject of many publications and reviews. Based on these studies, the population at risk, i.e., the critical receptor, in the United States has been identified as the urban child of one to six years of age (Buckley, et al., 1973). However, neurological damage may have actually occurred at an earlier period with the effects first appearing at a later stage of development. In addition, these effects may be too subtle to be easily recognized since they may gradually progress with growth and maturation.

Clinical evidence indicates that young children are much more likely to suffer from the effects of lead poisoning than adults. Also, children tend to become exposed to and absorb greater quantities of lead than do adults,

especially in the inner cities and older urban areas. This is probably due to lead-contaminated dust predominantly originating from automobile exhaust (Sayre, et al., in press) and from lead-contaminated household items such as paint chips. Further, there is evidence indicating toxic symptoms appear at lower dosage levels and that absorption rates are higher in children than in adults (Alexander, et al., 1973.)

There are about 17 million children, aged six or less in the United States. The National Bureau of Standards (Gilsinn, 1972) has estimated that 2.5 million of these children have high risk of lead poisoning and that over 600,000 of these children now have elevated lead levels in the blood (over 40 μg/100 ml). This study confirms that "pediatric lead poisoning is a major urban health problem in this country. The effects of this, in terms of the number of children suffering permanent brain damage or damage to other organs, is not known. How many of the children who have only slightly elevated blood-lead levels will later exhibit learning or social difficulties is not known."

Another estimate of children with high blood-lead levels is the "Lead Model Case Study" by Lutz, et al., (1970) which states that one-eighth of the United States population lives in substandard housing in metropolitan, ghetto areas. There are 10 million children in age groups 0.5 to 3.5 years, with 1.25 million children living in high risk areas. It was estimated that 250,000 young children (up to 3.5 years) have high blood-lead levels, and 1,900 children suffer permanent brain damage, each with an approximate $250,000 cost to society during their lifetimes.

At any rate, young children constitute, by far, the largest group of clinical lead poisoning cases--especially in high density urban areas and near heavily traveled streets and highways. This may be largely due to their much greater propensity to touch, mouth, and injest just about everything available in their environment.

While the acute effects of lead uptake are a matter of routine clinical observation and treatment, the precise effects of chronic exposures to lead at various exposure levels, rates, and modes are unknown. This is true for all age groups. A comprehensive research monitoring program should also take this into account.

Studies available on infants and children concerning the respiratory dose and pulmonary retention of lead are not comparable to those performed with adults. Knelson (1974) and King (1971) have reviewed information which pertains to this problem. The current state of knowledge permits only an indirect estimate of the inhalation of airborne lead. This can be done by using the data based on metabolic requirements for oxygen, and hence for air. Using basal metabolic requirements, the daily volume of air can be estimated. To this should be added an allowance for increased metabolic requirements due to infection, fever, and physical activity. For normal activity and growth, the basal values should be multiplied by a factor of 1.35, and, in the extreme case, by a factor of 2.0. Metabolic activity and requirements for infants and children can be compared with adults on a surface area basis.

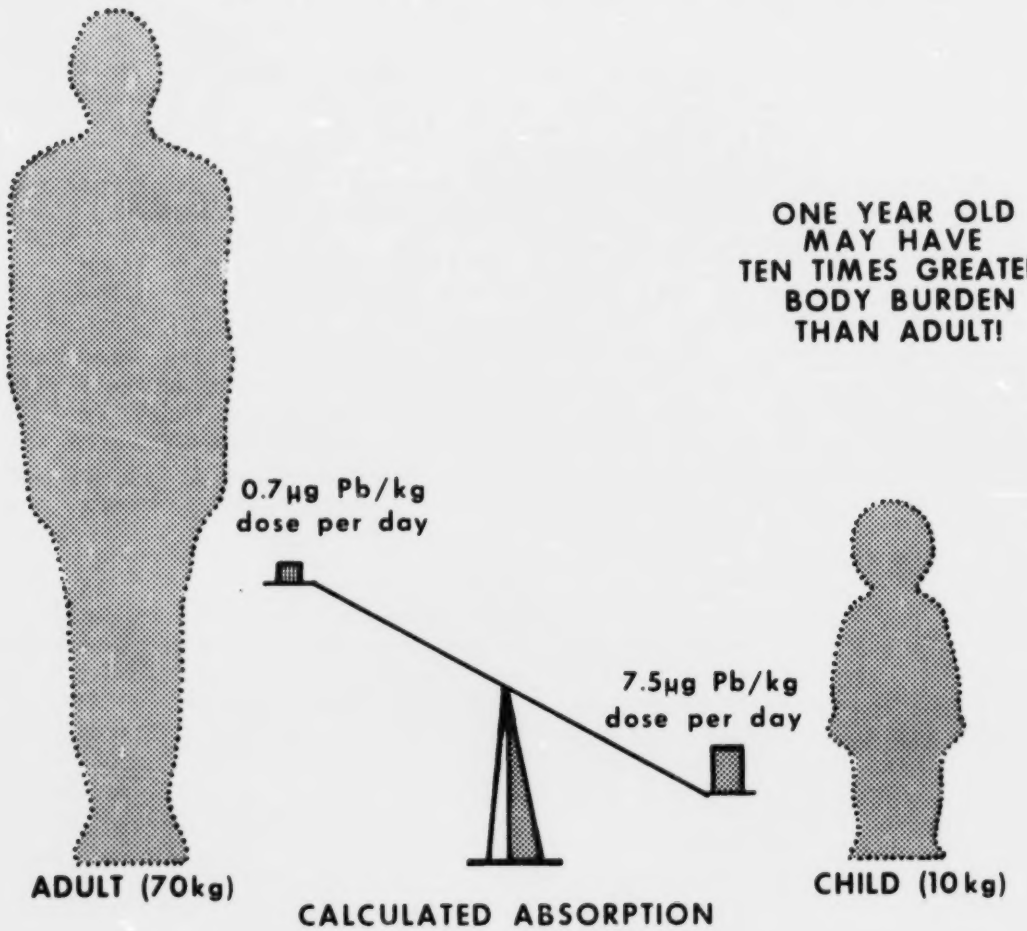
No statement can be made at present about such factors as particle size and its relation to the assimilation of lead by the respiratory route. It is important, however, to know the particle size distribution of lead, particularly the distribution below the two-micrometer level at from 1 to 3 feet above the surface. Measurements at this level are important in estimating respiratory exposures and doses in infants and young children. Although it is generally considered that there are no significant concentrations of organic lead compounds in the vapor form, some attention should be given to this if pilot studies show that there is a significant concentration. In this case, the different absorption factors for vaporized lead would have to be calculated.

Studies of the alimentary intake of lead in adults generally estimate the retention by this route to be up to about 10%.

For estimating alimentary intake in healthy children, the balance studies of Alexander, et al., (1973) are available; however, this study is limited to a short-term study in 11 subjects from 3 months to 8-1/2 years of age. These authors calculate an average daily intake of 10.6±4.0 µg Pb/kg body weight per day. Calculations were made for adults and children based on all of the data compiled during the workshop (Alexander, et al., 1973; Karhausen, 1973; National Academy of Sciences, 1972; Knelson, 1974). This more recent calculation indicates a daily absorbed intake of 7.5 µg/kg per day for children of 10 kg weight. (See Figure 1 for comparison with adult.)

Retention of lead in the body is partly dependent on the levels already existing. In small children the calculated retention was 2.03±1.4 µg Pb/kg/day or an average of 18% of the daily intake (Alexander, et al., 1973). These data, while preliminary, indicate that children take in less lead from air, water, and food than adults, and they absorb more in the intestine. Taking into account body weight, the resulting body burden of lead calculated for the child (Figure 1) is about ten times more per kilogram of body weight than for adults. This calculation does not include the additional exposure to lead children are subjected to from such sources as street and house dust which may be critical sources in some populations of children. It should be noted that the numerous studies showing that the concentration of lead in the blood of children is essentially the same as the concentration in adults does not constitute a rebuttal or counter-example to the above calculations. If a child rapidly stores lead in his soft tissues and bone, the blood-lead level will remain low even though he is absorbing large amounts and the tissue levels of lead are reaching toxic concentrations.

The whole blood-lead concentration is the most useful and accessible index of the level of lead in the exchangeable and potentially toxic pool within the body. Although individual measurements of whole blood-lead concentrations are not specifically related to adverse health effects, several ranges in blood-lead concentration can be related to varying degrees of risk of adverse health effects. The following broad ranges are recognized in adults, based on all available literature cited in the bibliography. The ranges for children are less well established, but seem to be similar except that they are somewhat displaced downward, i.e., the thresholds for the



CALCULATED ABSORPTION

Daily Intake	Lead Absorbed	Daily Intake	Lead Absorbed
Food - 300 µg	30 µg (10%)	Food - 130 µg	65 µg (50%)
Water - 20 µg	2 µg (10%)	Water- 10 µg	5 µg (50%)
Air - 2.5 µg (23 m^3)	21 µg (37%)	Air - 2.5 µg (6 m^3)	5.5 µg (37%)
TOTAL	±53 µgPb	TOTAL	±75.5 µgPb

$$\frac{53\ \mu gPb}{70\ kg\ man} = 0.7\ \mu gPb/kg\ dose\ per\ day\ to\ man$$

$$\frac{75\ \mu gPb}{10\ kg\ child} = 7.5\ \mu gPb/kg\ dose\ per\ day\ to\ child$$

Figure 1. Calculated Lead Body Burden Comparison Between Adult and Child

effects discussed below are somewhat lower in children. There are insufficient data available at this time to establish ranges for infants (less than 12 months of age).

The major evidence concerning ranges was derived from the interpretation of metabolic studies and Ca-EDTA mobilization data (National Academy of Sciences, 1972; Lutz, et al., 1970).

Range 1 (10-40 µg Pb/100 ml)

Studies in groups of normal healthy persons, primarily adults, show mean blood-lead concentrations between 15-20±6 (1 s.d.) µg Pb/100 ml of whole blood. While in vitro assays of delta-aminolevulinic acid dehydratase (ALAD) activity show partial inhibition in such groups, there is no measureable effect on the level of the substrate, namely urine delta-aminolevulinic acid (ALA), in body fluids.

Range 2 (30-55 µg Pb/100 ml)

This is the apparent threshold zone for the first detectable increase in ALA excretion and free erythrocyte protoporphyrin (FEP) levels. Above this threshold zone, there is a linear relationship between increases in blood-lead concentrations and the logarithm of increases in the levels of heme precursors in the body fluid. Early metabolic warning signals, of metabolic disturbance, such as heme synthesis and proximal renal tubular function, are available. However, there are at present no known biochemical indices of disturbance in brain function.

Range 3 (50-80 µg Pb/100 ml)

If blood-lead levels in this range are sustained for periods of weeks or months, most individuals will show adverse metabolic effects on heme synthesis, and a few may show early mild symptoms or plumbism.

Range 4 (70-100 µg Pb/100 ml)

Sustained levels in this range are associated with increasing risks of serious clinical effects, both acute and chronic. In comparison with groups having blood-lead concentrations in the 10-30 µg Pb/100 ml range, the sustained body burden must be increased by a factor of approximately 10 in order to reach the 70-100 µg Pb/100 ml range.

An important finding is that while there is a relatively narrow but more or less comfortable range between present levels of lead in the average adult (25 µg/100 ml of blood) and the toxic level (70-80 µg/100 ml of blood), no such range exists for children (Gilsinn, 1972).

The urban population of children has an average level of lead in the blood of about 25 µg/100 ml of blood, but 28.9% are 40 or above, 12.7% are 50 or above , 6% are 60 or above, and 2.7% are 70 or above. This includes over 600,000 children with levels over 40 µg/100 ml (700,000 based on New York data) (Gilsinn, 1972).

Symptoms such as anemia, behavorial problems and physiological changes have been observed at 40 µg/100 ml and over of lead in the blood of children. It is known that the threshold of lead toxicity in young children is lower than that of adults; however, how much below 40 or above 40 µg is not known. The disconcerting facts are that many blood levels of children are high and we know that the toxicity levels are in this range, but we do not know exact threshold levels or the variation in sensitivity which could result in many children having toxic problems at low levels. Therefore, the range between present levels and toxic levels in children is uncomfortably close with no latitude for safety (Lin-Fu, 1973).

Some evidence indicates that young children have a lower tolerance to blood-lead concentrations than do older population groups (Lin-Fu, 1973). In considering receptor levels of the monitored pollutant (lead), the vulnerability of developing brain tissue requires particular attention. Unlike other tissues which have an inherent ability to regenerate themselves, brain tissue is unique in its "once only opportunity to grow properly." Information heightening this critical concern is that the concentration of lead in the brain sharply increases only during the first two decades of life (Zaworski and Oyasu, 1973). Maximum rates of growth occur in the human prior to one year of age, are largely complete by 30 months of age, and appear to be greatest between the last eight weeks in utero and the first 10 months following birth. There may be another possible period of brain vulnerability - the 10th to the 18th gestational week of neuronal multiplication. This emphasizes the need to consider the pregnant woman in a pollutant monitoring model. This period of increased susceptibility may be associated with a lower tolerance for lead than is apparent in older age groups.

Autopsy data and metabolic studies (Rabinowitz and Wetherill, 1973) show that lead in the body is partitioned into two major compartments: (1) a small but rapidly exchangeable compartment consisting of blood, soft tissues, and the rapidly exchangeable fraction of bone mineral; and (2) a much larger but slowly exchangeable fraction tightly bound in bone and teeth. Autopsy data show that the bony fraction increases with age in members of industrialized and urbanized societies, but that soft-tissue lead concentrations show little apparent increase with age. A three-city study (U.S. Public Health Service, 1965) indicated that blood-lead concentrations do not show any measurable increase with age. The rapidly exchangeable pool is similar to, if not identical with, the chelatable fraction (or the metabolically active fraction of lead in the body. There are no documented clinical instances in recent years of toxicity related to the sudden mobilization of the bony stores. Experimental observations are not helpful in this regard as they do not closely simulate the normal human experience. Increasing concentrations of lead in the exchangeable pool are associated with the known adverse health effects of lead (Chisolm, 1971).

Experimental observations and clinical observations suggest that free erythrocyte protoporphyrin (FEP) measurements reflect sustained changes in soft-tissue lead levels.

Biological variables such as nutritional, genetic, metabolic, and hormonal factors (about which there is little information, but which could modify the absorption and distribution of lead) do not allow a more precise statement regarding blood-lead concentrations in various groups of people.

Blood-lead concentrations can be affected in the short-term by isolated spikes in assimilation due to factors like the ingestion of lead-containing paint chips or dusts. This can cause a transitory increase in the blood-lead concentration. Conversely, chelation therapy can temporarily depress the blood-lead concentration. However, the single, massive ingestion of lead is not associated with known severe toxicity (U.S. Public Health Service, 1971).

It is essential to measure lead directly in the receptor to determine the uptake resulting from environmental exposure sources. The available data (National Academy of Sciences, 1972) strongly suggest that measurements of the concentrations of lead in whole blood and the metabolite, erythrocyte protoporphyrin, are among the most sensitive indicators of changes of lead concentrations in the exchangeable pool which is responsible for lead's adverse effects. Among the various practical options for sampling, this is also the most available one. Random blood samples collected from children in selected environmental areas might give a fairly reliable indication of average exposure/effect relationships in these areas if the random samples are numerous enough to be representative, whereby unusual variances would cancel each other.

The methodology used for sampling and analyzing blood for lead is critical. Reliable results depend on stringent quality control procedures. Under good conditions, sampling and analysis errors on the order of ±5 μg Pb/100 ml (1.0 s.d.) should be anticipated in field studies.

While the blood-lead level is one measure of assimilation by the receptor, other types of data should not be disregarded in a monitoring system.

Teeth represent a host tissue in which the dentine is reflective of prior exposure over a non-specified length of time. It may be possible to utilize the lead-210 content of this tissue in order to determine the residence time of lead in the tissue. Levels in teeth do correlate with exposure but not to blood lead, and they normally decrease markedly at about age 10 in the human. While dentine-lead levels have been shown to correlate to exposure levels (Needlemann, et al., 1973), the information concerning any possible relationship to adverse health effects is insufficient at this time. In one study (Needlemann, et al., 1974), which included both analysis of teeth and blood-lead concentrations, a relationship between late health effects was found to be correlated with prior blood-lead concentrations, but not with tooth dentine levels.

Hair may afford a method of evaluating community exposures and changes therein, particularly exposures to airborne pollutants. The ready availability of this tissue would initially suggest that it is an ideal tissue for receptor monitoring. However, variations in hair growth rate, area of the body from

which obtained, hair color, racial background, and texture must be considered (Schroeder and Nason, 1969). Present methodologic difficulties are a strong deterrent to effective usage of hair as a primary receptor monitoring tool. A pilot study to assess possible relationships between hair-lead levels, blood-lead levels, and adverse health effects may yield meaningful data and facilitate the interpretation of hair-lead levels.

Fingernails or toenails are also readily accessible and should be considered in the design of a monitoring system. However, those measurements are subject to the same concerns that apply to hair.

As an available metabolic product, sweat should be considered for measurement in a biologic monitoring system. However, data for lead evaluated in several normal, white, male adults shows that the concentration of lead in activity-induced sweating is approximately 15 parts per billion, or 15 µg Pb/liter (Rabinowitz, 1974), and may vary depending upon the degree of active sweating; i.e., hypermetabolic versus passive or basal sweating level. Sweat may not be useful for monitoring purposes because of the low lead concentrations, the difficulty of obtaining sufficient uncontaminated samples, and the lack of quality control in accepted analytical methodology.

Saliva, like sweat, is a substance which one might include in a monitoring system. This material contains approximately 20 parts per billion of lead, or 20 µg Pb/liter, in healthy, white, male adults and apparently is in rapid exchange with blood lead (Rabinowitz, 1974). Significant deterrents to its usage in a large-scale monitoring approach are the difficulties inherent in obtaining adequate samples which are free of contamination by inhaled or ingested lead. These difficulties would be especially marked in young children.

A well-rounded biological lead monitoring research approach would probably include at least three basic analyses of blood samples collected with random or selective sampling techniques. These three analyses are:

(a) Whole Blood-Lead Concentration. This analysis gives a reliable indication of the amount of lead available in the exchangeable body pool, and allows some evaluation of its relation to general, and possibly specific, environmental lead exposure conditions.

(b) Free Erythrocyte Protoporphyrin (FEP). This is a sensitive method for measuring metabolic tissue effects from increased lead absorption levels, and may indicate the degree of possible permanent damage to the body.

(c) Hematocrit. This is a simple way to determine the possible relationship between iron deficiency and increased lead absorption.

Information of this type is already being obtained through childhood lead poisoning screening programs, and data are being tabulated by the Communicable Disease Center, Bureau of State Services, Division of Environmental Health Services, Atlanta, Georgia. Semi-micro techniques for both blood lead and erythrocyte protoporphyrin have recently been developed. Such sampling might

be rather easily extended to infants and pre-natal programs. For the sampling of children within this age range not covered under Medicare, it may be advisable to approach the American Academy of Pediatrics (Evanston, Illinois). With suitable administrative arrangements, such data could be made available to an integrated monitoring system.

Epidemiologic data and experimental observations suggest that women in the childbearing age range (especially during pregnancy), neonates, infants, and young children should be monitored. Sampling could possibly be made available to other groups through health maintenance organizations, similar pre-paid medical group plans, the armed forces, etc. If accessible, these groups should also be included.

Exploration of the utility of hair and shed deciduous teeth on a pilot basis should be considered. However, one should take into account the fact that such samples are not easy to collect and present considerable problems in both analysis and interpretation. The aim of such a pilot study should be to determine correlations between teeth and hair and nails, on the one hand, and blood-lead levels and indices of toxicity on the other hand.

If autopsy tissues can be obtained, they would be invaluable to increase what is currently a very limited data base with regard to tissue-lead levels in fetuses, infants, and children.

There is a great need for more information on lead metabolism in fetuses, infants, and young children. Not only with regard to dietary intake, but also concerning respiratory intake, and basic respiratory physiology in young children themselves. For this purpose, the feasibility of using the naturally occurring isotopes such as lead-210 or lead-204 as a marker deserves serious consideration. When such research is completed, the results will be useful in updating monitoring systems that are currently being proposed.

Adequate provisions for the protection of an individual's confidentiality must be considered in the design of programs involving human monitoring.

The most critical adverse effect of lead toxicity is irreparable damage to the brain and nerves. Although it has not been established that the brain is most vulnerable to lead during its growth and neuronal multiplication phases, this may be the case. If, indeed, the brain is most susceptible to lead during these phases, the situation may be associated with a lower tolerance for lead in children than is apparent in older age groups. Much more information is needed in this area.

Whole blood-lead concentration is the most useful and accessible index of the exchangeable and potentially toxic pool within the body. Although individual measurements of whole blood-lead concentrations are not specifically related to adverse health effects, several concentration ranges can be related to varying degrees of risk of adverse health effects. The threshold zone, derived from the interpretation of metabolic studies in adults and calcium-ethylenediaminetetraacetic acid (Ca-EDTA) mobilization data, appears to range between 30 and 55 μg Pb/100 ml of whole blood. The duration of exposure and

lead concentration levels in whole blood are related to the appearance of effects and to accurate dose calculation. Biological variables such as nutritional, genetic, metabolic, and hormonal factors probably modify the absorption and distribution of lead; however, little information is available in these areas.

While whole blood-lead concentration is the most accessible and valuable biological monitoring index, other media should not be disregarded in a comprehensive monitoring program. Teeth, for instance, may represent a host tissue which is reflective of prior doses over a non-specified length of time. Also, hair may afford a method of evaluating community exposure trends. The information obtainable from random samples of excreta and bodily secretions is deemed of negligible value for monitoring purposes.

CRITICAL TRANSPORT PATHWAYS AND PORTALS OF ENTRY

Once sound integrated monitoring system concepts are developed and applied to surveillance of environmental lead, as well as other pollutants, it will be possible to further delineate areas of concern, to provide reliable trend and research data, and to establish efficacious and economical control measures.

Dispersion of lead into the environment is relatively broad and diffuse. However, dispersal from the critical source to the critical receptor involves specific pathways which must be quantified and accurately monitored. To this must be added the general background level of lead.

The detailed exposure pathways contributing to lead burdens in a receptor are shown in Figures 2 and 3. An objective of this study was to quantitate these pathways and, thus, to determine total exposure and its variability among individuals and populations. Additionally, the relations between exposure, dose, uptake, and effects were assessed. The diagram in Figure 4 illustrates the pathways of lead within a critical receptor. These pathways were assessed to determine the best indices of body lead burdens.

In spite of the availability of an impressive information base (Lutz, et al., 1970), the pathways leading to and within a critical receptor, the urban child, cannot be accurately quantitated without additional substantial investments in research and monitoring. One result of this lack of quantification is that most current review documents tend to overestimate the contributions of leaded paint chips and to underestimate the importance of airborne lead which continually settles out on all surfaces and is available for resuspension and subsequent inhalation and ingestion.

The major sources of airborne respirable dust particles containing lead are transportation, industry, energy production, and incineration. The particle size distribution, after gravitational and atmospheric interaction, is believed to be approximately a logarithmic normal distribution for all sources. Of these sources, motor vehicles using leaded gasolines contribute by far the greatest quantities of all airborne lead particles (Buckley, et al., 1973).

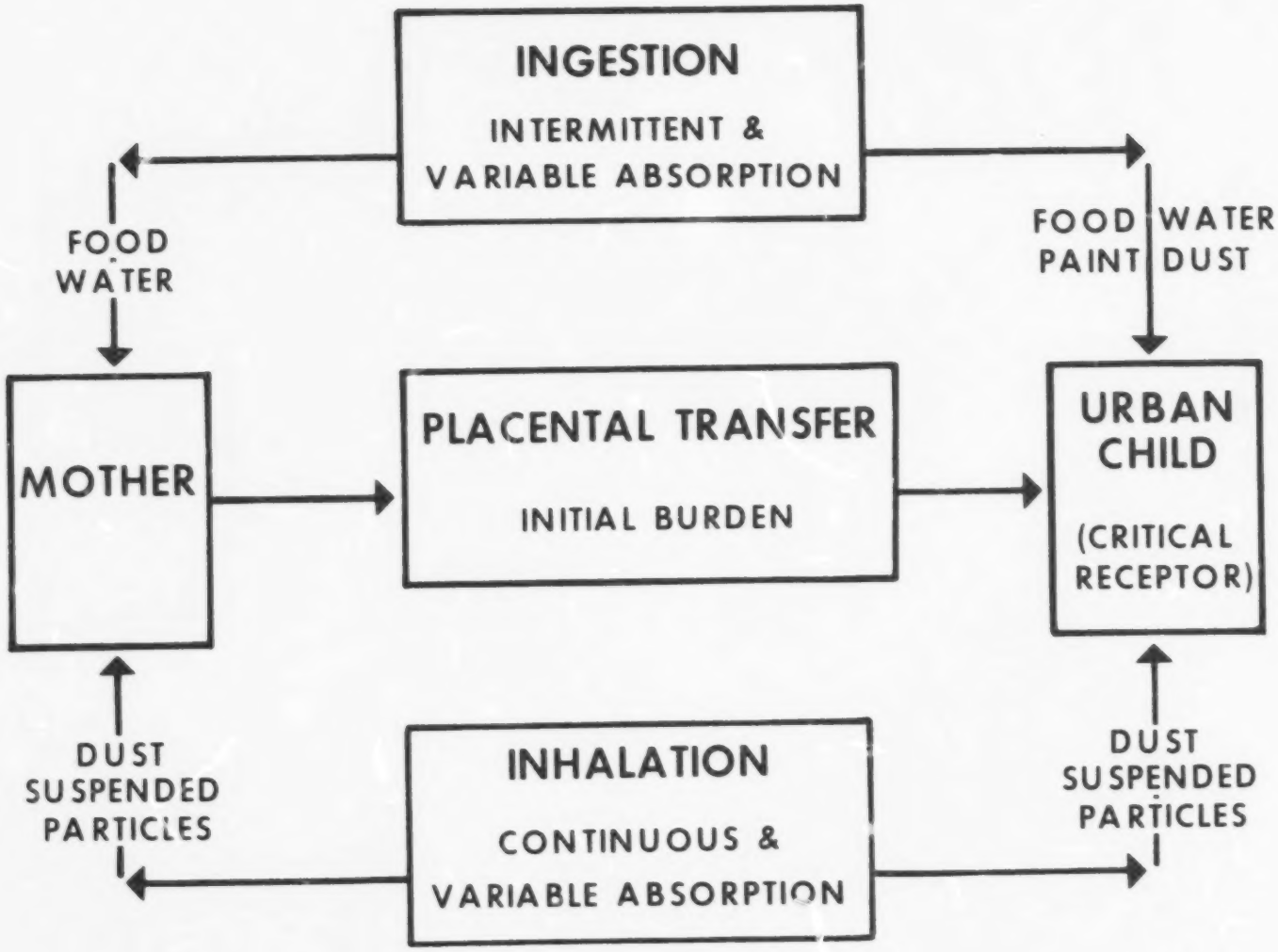


Figure 2. Major Pathways Affecting Lead Burden in Critical Receptor

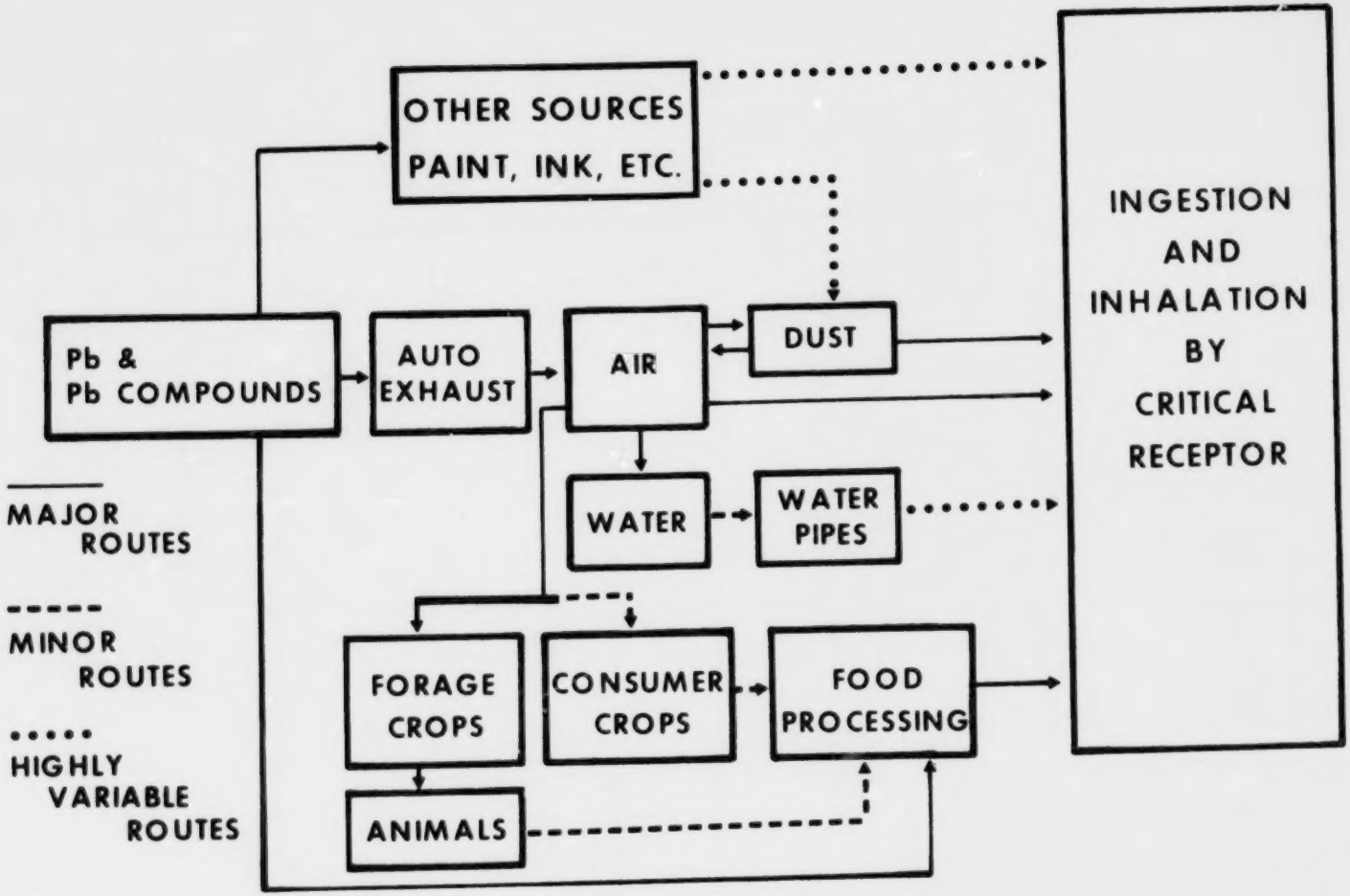


Figure 3. Lead Exposure Pathways to Critical Receptor

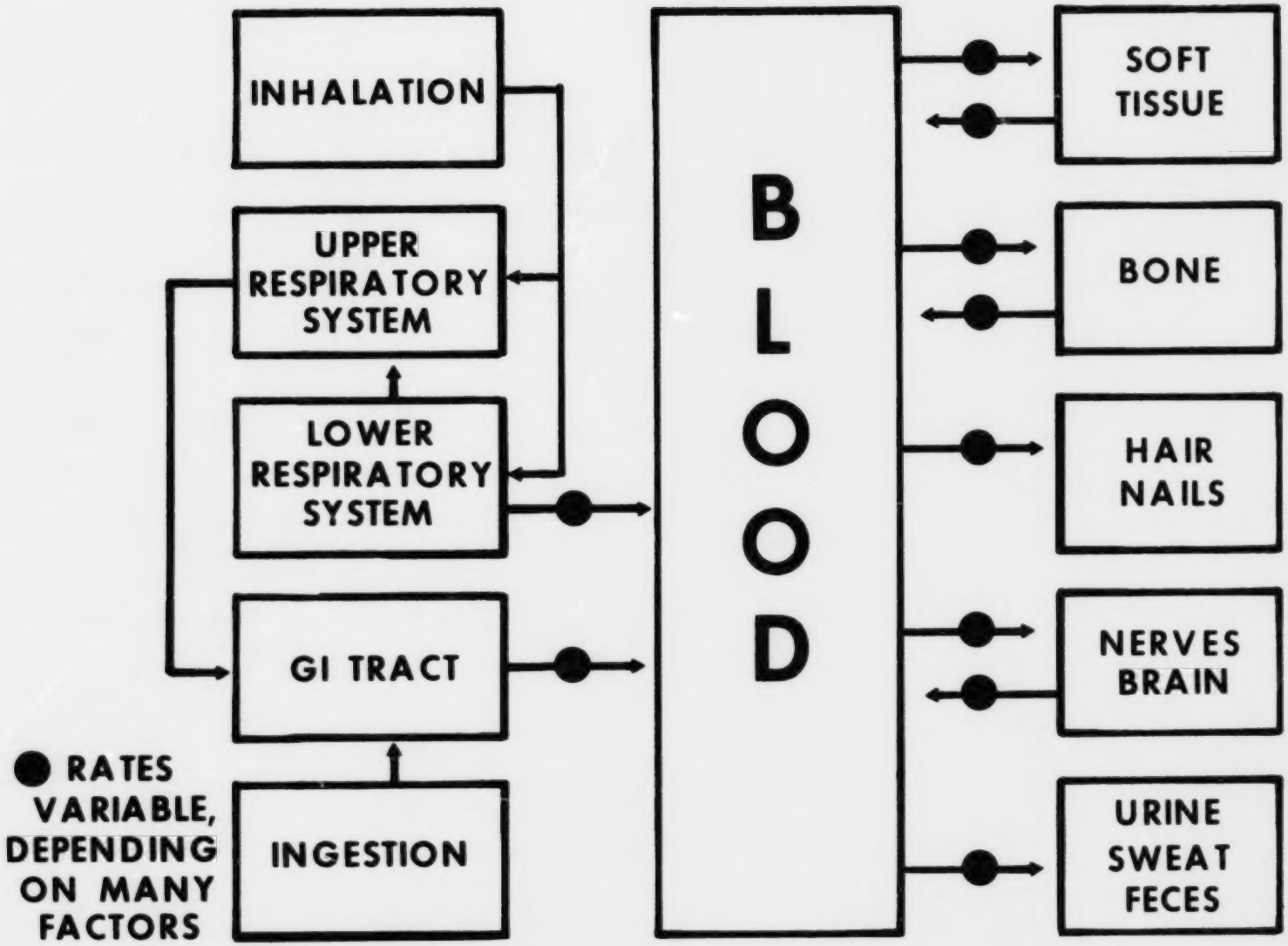


Figure 4. Pathways of Lead in Critical Receptor

The airborne concentration of lead at any specified location can be determined by summing the relative contributions from mobile and stationary sources, the geophysical background and the atmospheric lead originating from other cities. Meteorological conditions, proximity to significant stationary sources, and the relation between traffic volume and distance from the critical receptor population groups are important monitoring considerations.

The largest source of routine ambient air-lead data in the United States is the National Air Surveillance Network (NASN). These stations, which are located both in urban and non-urban places, provide a general indication of overall "average" urban lead concentrations. In addition, many specialized measurement studies have been undertaken very near important lead sources, such as highways and intersections, and these are reported in the literature.

The arithmetic mean air-lead concentration in the United States (U.S. Public Health Service, 1965) (from 1960 to 1965) was 0.79 $\mu g/m^3$, using data from NASN high-volume samplers. During this period of time, the highest single air-lead concentration in any city was 8.6 $\mu g/m^3$.

Many investigators, such as Larsen (1969), hold that the concentration in air of pollutants in general follows a lognormal distribution. (It should be noted that the arithmetic mean is not an appropriate statistical measure of central tendency for the lognormal distribution; the geometric mean should be used).

Table 1 gives typical annual mean air-lead concentrations from several cities. Typical outlying concentrations are on the order of 1 $\mu g/m^3$; concentrations in the more central areas are generally in the range of 2 to 4 $\mu g/m^3$. The table also shows examples of the higher air-lead concentrations that are encountered very close to sources. Concentrations measured near traffic and on sidewalks range between 8 and 30 $\mu g/m^3$, with some values as high as 54 $\mu g/m^3$ for the morning rush hours. (National Acadamey of Sciences 1972).

Table 1. ATMOSPHERIC LEAD CONCENTRATIONS

Of Cities	Annual Mean
Philadelphia, suburbs	1 $\mu g/m^3$
Philadelphia, downtown	3 $\mu g/m^3$
Cincinnati, suburbs	1 $\mu g/m^3$
Cincinnati, downtown	2 $\mu g/m^3$
Los Angeles, suburbs	2 $\mu g/m^3$
Los Angeles, downtown	3 $\mu g/m^3$

Near Roadways	Range of Annual Means
Los Angeles, freeway (a.m. rush hour)	26.9-54.3 $\mu g/m^3$
Los Angeles, freeway (mid-day)	16.6-31.1 $\mu g/m^3$
Los Angeles, downtown traffic (a.m. rush hour)	19.1-29.9 $\mu g/m^3$
Los Angeles, downtown traffic (mid-day)	8.4-12.2 $\mu g/m^3$
Pasadena, downtown, sidewalk (mid-day)	8.6-14.6 $\mu g/m^3$

It should be noted that all the data are not strictly comparable because the sample durations (i.e., averaging times) are often different, or worse, are not stated at all. Furthermore, the exact distance and height at which the measurements are made are often unspecified. However, some data do exist in the literature on concentration "profiles" as a function of distance from sources. Elaboration on this subject is beyond the scope of this review; however, consideration of these factors is essential in the design of monitoring networks.

It can be concluded from the NASN data previously cited that ambient air urban-background lead concentrations are in the range of 1 to 3 $\mu g/m^3$ in the majority of urban areas (annual average), with concentrations within 100 meters of streets with heavy traffic loads usually ranging from 8 $\mu g/m^3$ to about 30 $\mu g/m^3$.

Fairly extensive data are available on the size distribution of atmospheric lead-containing particulates (Robinson and Ludwig, 1964). The distribution is generally approximated as log-normal; i.e., the logarithm of aerodynamic particle size is normally distributed. A typical cumulative plot of particle size on log-probability paper is shown in Figure 5. Note that over 90% of the mass of airborne lead particles are under one micrometer in size. Lead particles of this size distribution can readily be respired and thereby entered into the bloodstream.

Similar data on the size distribution of the larger diameter particles which settle out of the atmosphere are unavailable. These particles contribute to the lead burden of the dust encountered by children and vary widely in size due to the effects of control systems on sources such as smelters, refining and metallurgical processes, and on the internal combustion engine and its exhaust system. However, since particulates of very large diameter (greater than 25 microns) and intermediate diameter (10-25 micrometers) can be inspired and/or ingested by the critical receptor, they are of high concern.

Settled dust originates from the same sources as airborne respirable dust particles - namely, transportation, industry, energy production, and incineration. Here again the leaded gasoline used in motor vehicles contributes most of the lead in dust in most residential areas. However, there are important exceptions. For example, the El Paso, Texas, lead smelter emissions dwarf the contributions of lead dusts by automobiles and lead-based paints in some El Paso residential areas (Landrigan, et al., 1974).

Lead in settled dust and dirt also contributes to increased blood-lead levels through the inhalation of resuspended dust and through ingestion primarily by children. Although exposure to dirt and dust contaminated with lead from automobile exhaust, alone, has not been shown to be responsible for cases of overt lead poisoning, it has been related to high blood-lead levels in children (Buckley, et al., 1973). There is also a good correlation between high blood-lead levels in children and household dust. The lead in dirt and dust on children's hands, and the consequent higher blood-lead levels also increase with nearness to heavily traveled streets and mid-

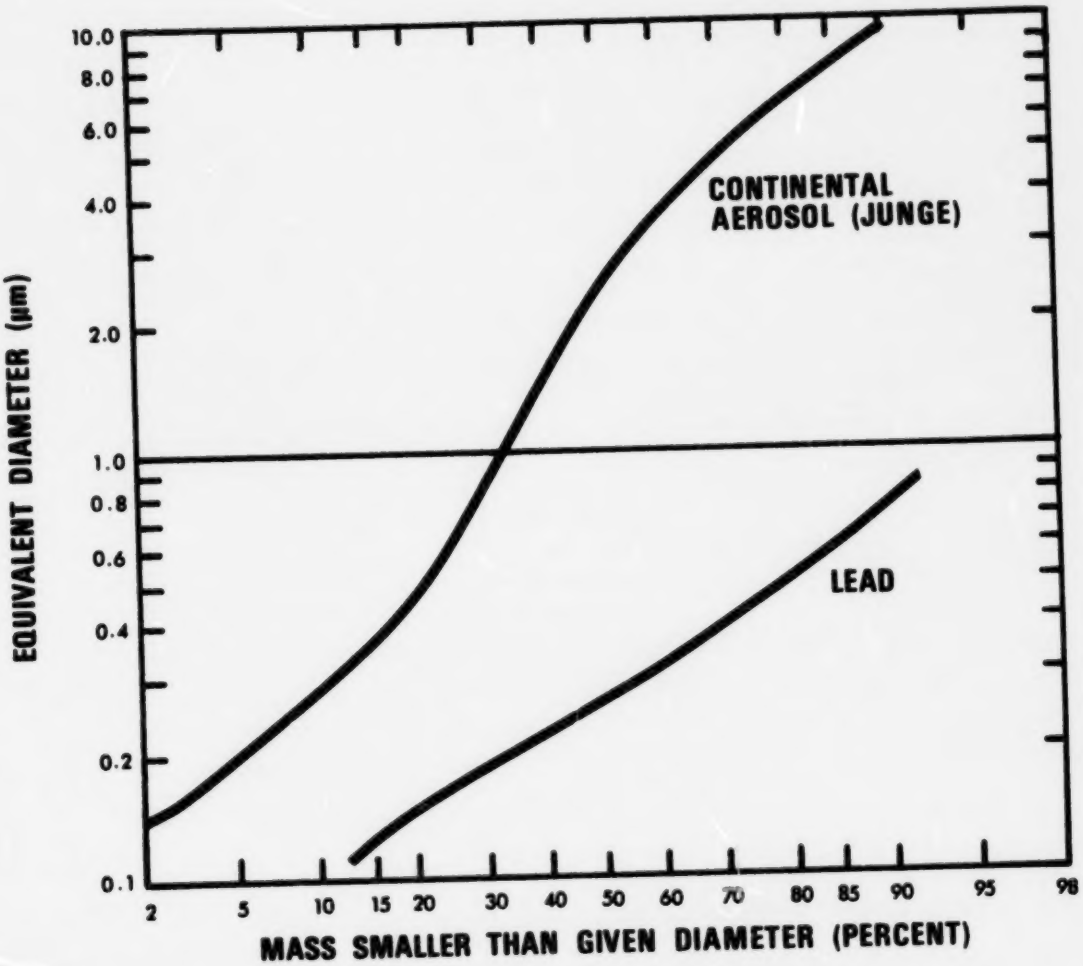


Figure 5. Average lead size distribution comparison, with Junge's model for continental aerosols on log-probability paper. (Tabular information adapted from Robinson and Ludwig, 1964.)

city areas, while adults in the same environment show substantially lower blood-lead concentrations than do children.

Recent surveys of several urban environments show that there are substantial concentrations of lead in the dust and dirt on and near heavily traveled roadways (see Table 2) and in residential areas (see Table 3). The fallout of airborne lead from automobile exhaust and that from other mobile and stationary sources creates a significant exposure hazard, especially for children. Dust and chips from the deterioration of leaded paints are also major contributors to interior and exterior residential lead pollution.

Table 2. LEAD DUST ON AND NEAR HEAVILY TRAVELED ROADWAYS

Location	Sampling Site	µg Pb/g	Authority
Washington, D.C.	Busy Intersection	12,820	Lin-Fu (1973)
	Many Sites	(4,000-8,000)	" " "
Chicago	Near Expressway	6,000	Calandra (1973)
Philadelphia	Near Expressway	3,000-8,000	Needleman, et al., (1973)
Brooklyn	Near Expressway	900-4,900	Lombardo (1973)
Boston	Near Expressway	1,000-2,500	Fritch and
Vermont	Near Expressway	500-700	Prival, (1972)
New York City	Near Expressway	2,000	Pinkerton, et al., (1973)
Detroit	Street Dust	966-1,213	Ter Haar, et al., (1973)
Philadelphia	Gutter (Low Exposure)	1,507 (270-2,626)	Needleman, et al., (1973)
	Gutter (High Exposure)	3,262 (280-8,201)	" " "
Various U.S. cities	Highways and Tunnels	10,000-20,000	Buckley, et al., (1973)
Netherlands	Heavily Traveled Roads	5,000	Rameau (1973)

Sayre, et al., (in press) considered house and hand dust as a potential source of lead exposure to young children. Their findings showed a strong relationship between lead concentrations found on household surfaces and those found on children's hands in the same households. No significant correlation could be made between the household dust-lead level and whether or not leaded paint was available in the household. In addition, they found that inner-city children have higher mean blood-lead values than suburban children, and the lead content of dusts collected from the houses and hands of inner city children is significantly higher.

Table 3. LEAD DUST IN RESIDENTIAL AREAS

Location	Sampling Site	µg Pb/g	Authority
Philadelphia	Classroom	2,000	Needleman, et al., (1973)
	Playground	3,000	
	Window Frames	1,750	
Boston and New York	House Dust	1,000-2,000	Needleman & Scanlon (1973)
Brattleboro, Vt.	In Home	500-900	Darrow & Schroeder (1973)
Birmingham, Eng.	In Home	5,000	Lombardo (1973)
New York City	Middle Class Residential	608-742	Pinkerton, et al., (1973)
El Paso Smelter	Smeltertown Dust at:		
	0-1 Miles	36,853 (2,800-103,750)	Landrigan, et al., (1974)
	1-2 Miles	2,726 (100-84,000)	"
	2-3 Miles	2,234 (100-29,386)	"
	>4 Miles	2,151 (200-22,700)	"
Philadelphia	Urban Industrial	3,855 (929-15,680)	Needleman, et al., (1974)
	Residential	614 (293-030)	"
	Suburban	830 (277-1,517)	"

Lead pollution at the soil surface level varies markedly, but it generally decreases with distance from central urban areas, roadways, smelters, power plants, painted buildings, and other sources of lead. The results of some recent studies documenting lead surface soil pollution are presented in Table 4.

Household paints containing more than 1% lead are very important contributors to clinical lead poisoning and elevated blood-lead levels in children. This is especially true for older deteriorating houses where the paint is chalking, flaking, and/or peeling, and young children between the ages of 6 months to 3 years are present. The pica habit of these young children, i.e., eating non-food items, accounts for the high ingestion rate of leaded-paint particles and the consequent doses.

Table 4. LEAD AT SOIL SURFACES

Location	Locality	µg Pb/g	Authority
El Paso smelter area	600 feet from smelter	3,457	Landrigan, et al., (1974)
Midwest city survey	Residential	1,636	Hardy (1971)
	Commercial	2,413	" "
	Industrial	1,512	" "
San Diego	City Park	194	National Academy of Sciences (1972)
San Francisco	City Park	560	
Los Angeles	City Park	3,357	
Adjacent to U.S. highways	Soil	160-540	Seeley, et al., (1972)
England	Soil	10,000	Barltrop & Strahlow (1973)
High Sierra mountains	Soil	9.2	Hirao & Patterson (1974)

Although the ingestion of lead based paint chips by young children is often the ostensible and/or actual cause of overt clinical lead poisoning, these paint chips may have served only as a trigger for the acute symptoms requiring immediate treatment. These clinical cases may not have occurred in the absence of pre-existing chronic high blood-lead concentrations originating from airborne and/or settled dusts. Paint chips are available to young children especially in older, deteriorating houses that are improperly maintained.

Despite the existence of a Health Code limiting the level of lead in household paint for over a decade, Finberg and Rosen (1973) showed that significant lead can be found in vinyl acrylic paints. Many of these paints contained from 4 to 6 micrograms of lead per gram of paint despite the fact that they were labelled for interior use.

Lead has been found in the majority of food items tested throughout the world. Several investigations were made to determine the lead content of typical diets for groups of adults and children. These diets were considered to be representative of the intake of the groups studied. An excellent, though unexpected, agreement was observed among the various groups' average reported values in spite of their culinary differences. Some of the typical

average daily intakes of lead which appeared in the literature are shown in Tables 5, 6, and 7.

Table 5. LEAD IN DIET OF ADULT MALES

Adults	Average µg Pb/day*	Authority
United Kingdom	274	Thompson (1971)
England	220	Monier-Williams (1950)
England (1,500 g)	200	Tolan & Elton (1973)
Japan	150	Imamura (1967)
United States	300	Kehoe (1961)
United States	300	Kehoe (1963)
United States	330	Patterson (1965)
United States	330	Schroeder & Balassa (1961)
United States	270	Parry-Howells (1971)
United States	100-500	Schroeder & Tipton (1968)
United States	300	Cholak & Bambach (1943)
United States	285	Harley (1970)
United States	300	Lewis (1966)
United States	258	Schroeder & Balassa (1961)
World Average	440	Karhausen (1973)

*Assumes 2,000 g of food per day (some British diets assume 1,500 g).

From a sampling of New York City diets in 1966 (Harley, 1970), an interesting comparison can be made of the concentrations of lead in fresh versus processed foods (Table 8). Note the large increases in lead concentrations when fruits and vegetables are processed and when flour is converted into bakery products. These increases in lead content during processing are the result of such factors as contributions of lead from the food handling machinery and from containers.

Table 6. DIET OF 15-20 YEAR OLD MALES
(Food and Drug Administration 1973 Unpublished Data)

Food Product	gms/day ingested	ppm lead in food	µg Pb per day ingested
Dairy (milk-cheese-eggs)	756	Trace	Trace
Meat (fish-beef-poultry)	290	0.015	4.4
Grain and Cereal	369	0.012	4.4
Potatoes (fried-boiled-chips)	204	0.004	0.8
Leafy Vegetables	59	0.054	3.3
Legume Vegetables	74	0.265	19.6
Root Vegetables	34	0.131	4.5
Garden Fruits (tomatoes and cucumbers)	88	0.108	9.5
Fruits	217	0.031	6.7
Oil-Fats-Shortening	52	0.15	0.8
Sugar-Adjuvants	82	0.008	0.7
Beverages (tea-coffee-soda)	697	0.004	2.8
TOTALS	2,922 gms/day	0.782 ppm Pb	57.4 µg Pb/day

Table 7. LEAD IN DIET OF CHILDREN

Age	µg Pb/day	Authority
Young Adult	165	Chisolm & Harrison (1956)
2 Year Old	133	Barltrop (1973)
Youth (10 kg)	120	Alexander, et al. (1973)
Youth (20 kg)	155	" " "
Youth (25 kg)	180	" " "
Youth (30 kg)	210	" " "
Italian Infant:		
4 Month Old	172	Lanzola, et al. (1973)
6 Month Old	224	" " "
9-12 Month Old	289	" " "
Infant	100	Kehoe, et al. (1933)
Infant	150	Barltrop & Killala (1967)
Infant	165	Chisolm & Harrison (1956)

Table 8. LEAD IN FRESH AND PROCESSED FOODS

Adapted from table by Harley (1970)
and from table by Tolan and Elton (1973)

Diet Category	Fresh mg/kg wet weight	Processed mg/kg wet weight
Vegetables	0.12	0.44
Fruit	0.07	0.25
Whole Grain Products	- - -	0.13
Flour	- - -	0.04
Macaroni	- - -	0.08
Rice	- - -	0.04
Bakery Products	- - -	0.39
Meat	0.42	- - -
Poultry	0.30	- - -
Eggs	0.22	- - -
Dairy Products	- - -	0.04
Shellfish	0.31	- - -
Fresh Fish	0.16	- - -
Carrots	0.02	0.22
Peas	0.02	0.21
Corn	0.01	1.22
Spinach	0.02	0.95
Beef	0.23	1.20
Cherries	0.05 (jars)	0.17 (cans)
Baby Foods	0.04 (jars)	0.24 (cans)

An on-going program in the Food and Drug Administration (FDA) currently measures lead levels in 41 major food commodities including fluid milk and nine baby foods. In Fiscal Year 1975 the FDA will conduct two additional total diet studies. One will focus on the lead content of foods consumed by the 6-month-old infant and the other will be concerned with the same problem in the two-year-old toddler. (The current FDA guideline for lead in evaporated milk is 0.5 ppm).

The analytical procedure which has been used to date by FDA (FDA, 1973) in obtaining lead data is based on atomic absorption. The FDA is readying an anodic stripping voltametry (ASV) procedure which will probably be used in future surveys involving heavy metals.

The Children's Hospital in Washington, D.C. is currently surveying the blood-lead levels of children in the inner city. In support of this survey, the United States Food and Drug Administration Baltimore Laboratory is analyzing the food sources of these children for lead.

The total diet survey conducted by the FDA indicates that the average daily intake of lead by a 15 to 20 year old male is 57.4 µg/day. Certain assumptions have been applied to the data used to calculate this value in an effort to compensate for inadequacies in the reported values. If one converts all values reported as "trace" to 0.09 µg Pb, on the assumption that 0.1 µg Pb would be the lowest reported value, then the average daily intake increases to 159 µg Pb/day. Furthermore, if all values reported as "zero" are assumed to have the finite value 0.05 µg Pb, then the average daily intake increases to 233 µg Pb/day. The conclusion based on the Fiscal Year 1973 data is that the average daily intake of lead in the food by a 15 to 20 year old male is between 57 and 233 µg/day.*

In a similar study, the Food and Drug Protectorate of Canada concludes that the lead intake from food is about 180 µg Pb per day for a young adult.

Although children ingest a lower total amount of lead, on a body weight basis (assuming 70 kg for the average man and 10 kg for the average one-year-old child), the amount absorbed for the child is greater, as discussed previously.

Drinking water, on the average, contributes very small amounts of lead to the body burden. Very few people drink water containing more than 50 µg Pb/liter. Problem areas would primarily involve lead plumbing and lead-glazed drinking vessels, particularly with acidic drinks. For monitoring purposes, water quality baselines should be established for potable water supplies just prior to distribution, and trends in suspended and dissolved lead should be evaluated. Pathways of lead to the critical receptor via water and other liquids are shown in Figures 6 and 7.

The analysis of more than 1,500 samples of raw surface waters at more than 100 locations across the United States from 1962 to 1967 indicated a range of dissolved or colloidal lead concentrations (i.e., those passing through a 0.45-µm millipore membrane filter) of between none detectable and 90 µg/liter. Only 23% of the samples yielded positive results. The arithmetic average was on the order of 10 µg Pb/liter. The average of the positive results was 23 µg Pb/liter (Kopp and Kroner, 1970). In the 1970

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*This information was provided in a personal communication from Mr. Paul Corneliusses of the FDA. The assumptions discussed above were impressed on the data in an attempt to counteract the effect of dilution resulting from the combination of several samples.

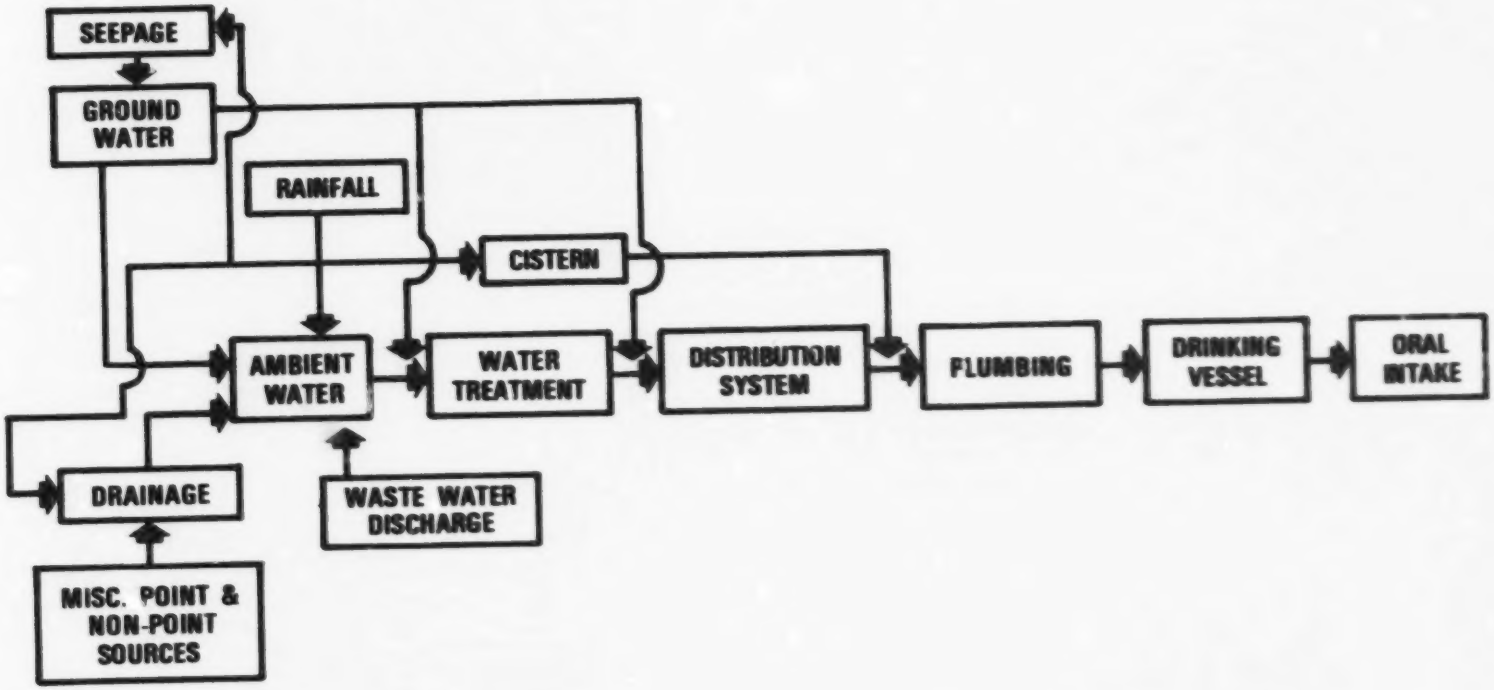

Figure 6. Pathways of Lead to a Receptor Via the Water Cycle

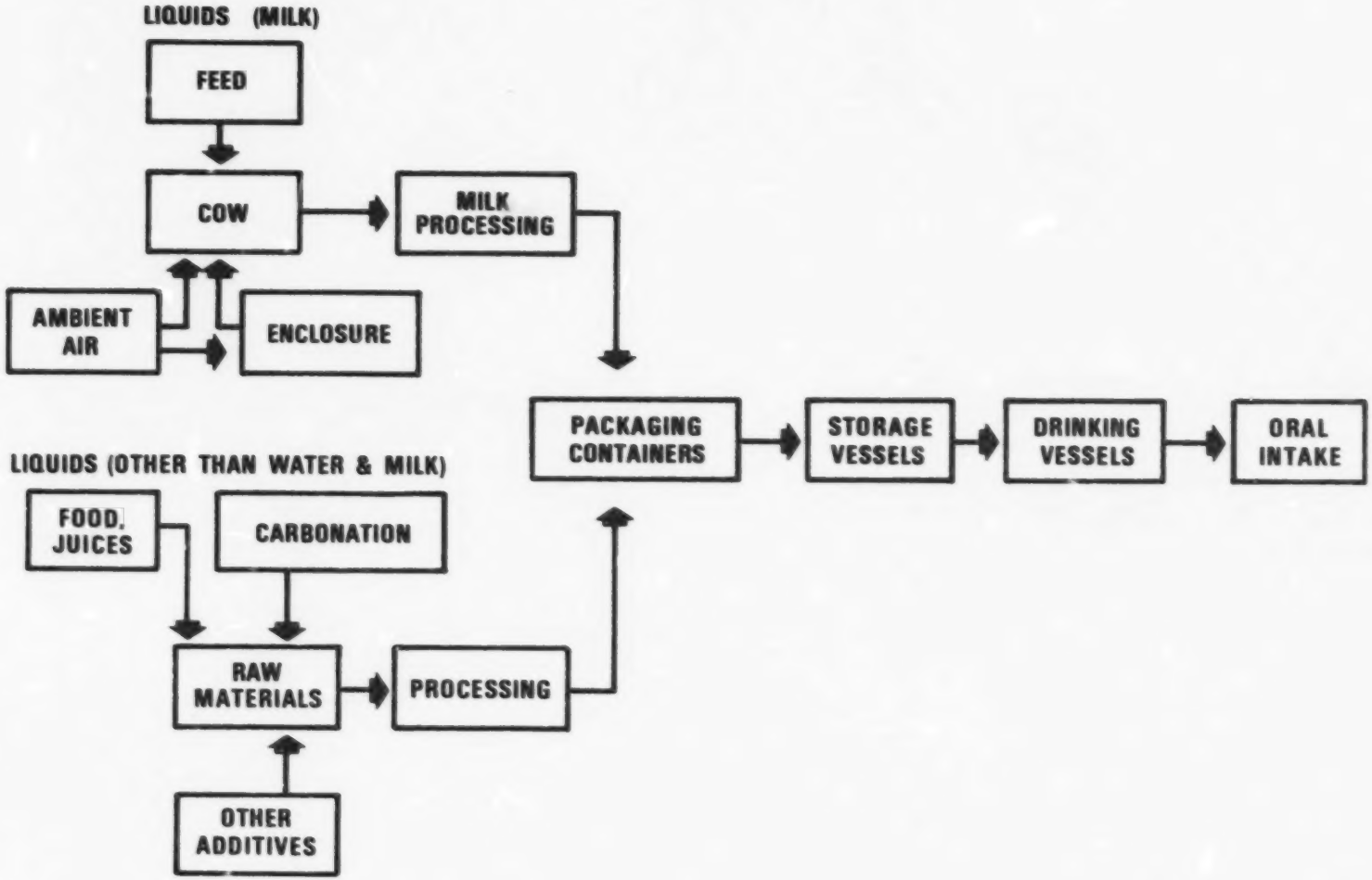

Figure 7. Pathways of Lead to a Receptor Via Liquids Other Than Water

United States Geological Survey analysis of 720 samples of raw surface waters across the United States, lead in the dissolved or colloidal form was found in 63% of them. The range in the positive samples was from 1 to greater than 50 µg/liter. Lead occurred widely in the 6 to 50 µg/liter range (USGS, 1970). In areas of limestone and galena, waters are known to contain lead in solution as high as 400 to 8,000 µg/liter (Kopp and Kroner, 1970). This is highly unusual, however.

Of 23 rainfall samples collected, concentrations of particulate material plus dissolved lead as high as 490 µg/liter were obtained. The lead concentrations increased with nearness to highways and other sources (Ettinger, 1965).

A range of none detected to 62 µg/liter of dissolved or colloidal Pb (the average was less than 10 µg/liter) was found in a 1962 survey of the 100 largest water supplies in the United States (Durfor and Becker, 1964). A range of none detected to 60 µg/liter of dissolved or colloidal lead was found in samples from 544 water supplies in the United States in 1973. The majority of values were less than 10 µg/liter (Buckley, et al., 1973).

Water standing in lead pipes exhibited as much as 920 µg/liter lead (Kehoe, 1961). No data are available on the lead contributed by water distribution systems other than that the interior of steel storage tanks is often painted with lead chromate paints.

Hardy (1965) reported that beer contains 100 µg/liter of lead and wine from 80 to 860 µg/liter. These values are supported by data presented by de Treville (1964) which show Cincinnati beer to range between 10 and 290 µg Pb/liter and wine between 50 and 1,510 µg Pb/liter. Some of the data cited in de Treville's report are given in Table 9.

Processed milks in 1972 and 1973 were found to deliver up to 100 µg Pb/day to toddlers. Fresh human milk and homogenized cow milk were found to contain no detectable lead (less than 0.5 µg/100 ml). Table 10 contains data on the concentration of lead in liquid milks (Lamm and Rosen, 1974).

Cases of lead poisoning in humans have resulted from use of acidic drinks with lead-glazed utensils. One case resulted after a patient had two years of habitual exposure to lead from drinking liquids with a pH of 2.7 from a glazed mug (Harris and Elsea, 1967). Tests were made of the lead contribution from a glazed mug when filled with chilled cola, with pH 2.7, for varying time periods. The cola before being poured in the mug had a lead concentration of 200 µg/liter. After two minutes, about 3,000 µg/liter of lead was leached from the mug; after two hours, the lead level was as high as 6,800 µg/liter. It was found that the lead contribution by lead-glazed drinking vessels varies greatly with: (1) the temperature at which the glaze was fired; (2) the time period during which the drink remained in the vessel; (3) the pH of the drink; and (4) the number of times the vessel had been used previously. As an instance of the influence of pH, it was found that no lead was detected in water that was retained in the same mugs which had contributed large concentrations to cola with a pH of 2.7 (Harris and Elsea, 1967).

Table 9. LEAD CONTENT OF VARIOUS BEVERAGES, IN MILLIGRAMS PER LITER
(Information from de Treveille, 1964)

Tea	
Tea (Ceylon), hand-picked (dry)	0.02
Tea leaves, import package lined with corroded lead foil (dry)	43.2
Tea (India), packed in tins	1.8
Tea (India), packed in lead foil	2.4
Beer	
Beer (Cincinnati)	0.01-0.29
Grape Juice and Wine	
Grape Juice	0.04-0.06
Wine	0.05-1.51
Liquors and Spirits	
Brandy (India)	0.05-0.06
Rum (India)	0.024-0.057
Dry Gin (India)	0
County spirit (India)	0.026
Whisky (India)	0.027
Aerated Waters	
Lemonade	0.004
Soda	0.004

Table 10. LEAD IN NEW YORK AREA MILK
(Information from Lamm and Rosen, 1974)

Type of Milk	Sources	Samples Analyzed	Mean ± 1 s.d. (µg Pb/100 ml)	Range (µg Pb/100 ml)
Evaporated (Skimmed)	2 Brands	11	6.0 ± 0.2	4-7
Evaporated (Whole)	2 Brands	15	11.0 ± 1.1	4-22
Infant formula:				
Concentrate	2 Brands	6	8.3 ± 0.9	4-12
Ready to serve	2 Brands	30	3.3 ± 0.4	0-8
Homogenized cow milk	3 Brands	19	<0.50*	
Human Breast Milk	10 Women		<0.50*	

*Limit of detection of non-flame atomic absorption spectrometry method used in this study.

A report demonstrated that pottery is capable of imparting as much as 7600 µg/liter of lead after an 8-hour acetic acid exposure (Stek, 1974). In another study, twenty-five percent of the earthenware gave 100 µg/liter lead in a standard test (Harris and Elsea, 1967). In addition, a plastic tank used to store soft water for 18 months imparted 500 µg/liter of Pb to the water. No lead was imparted to hard water by the same tank (Chancellor, 1960).

The pesticide, lead arsenate, contributes in some places to the contamination of our air, water, and food supplies via spray drift, fugitive dust, agricultural run-off, and by systemic incorporation in and by residues on 23 common fruits and vegetables. This source is not as significant as it once was due to a decline in its use. However, residential districts near agricultural areas using lead arsenate, or on land previously used for orchard crops, should be monitored. Also, market-basket samplings should be made to identify potentially harmful lead concentrations.

The domestic production of lead arsenate in 1971 was 3,100 tons,* up 48% from 1970. The 1971 production was down 14% from the average production of the previous five years, i.e., 3,550 tons. In 1971, the domestic disappearance (contributions to environmental pollution) was 2,550 tons. This represents a decrease of 29% over the average of the previous five years (Fowler, 1973).

In 1972 domestic production of lead arsenate was 2,791 tons, down 9.5% from 1971; however, domestic disappearance was 2,512 tons, up 21% from 1971 (Fowler, 1974).

A tolerance level of 7 parts per million combined lead has been established for lead arsenate on the following 28 crops: apples, apricots, asparagus, avocados, blackberries, blueberries, boysenberries, celery, cherries, cranberries, currants, dewberries, eggplant, gooseberries, grapes, loganberries, mangoes, nectarines, peaches, pears, pepper, plums, prunes, quinces, raspberries, strawberries, tomatoes, and youngberries; and 1 part per million on citrus crops (Code of Federal Regulations).

The Office of Pesticide Programs (OPP), EPA, operates a soils monitoring program. The samples include both inner-city and suburban soils. These samples will be analyzed for lead and will continue to be available for reanalysis in a soil bank maintained by OPP.

All golf courses in the northeast and midwest were identified by the OPP. A random sampling of the soil from the greens and fairways was made and the following lead levels were found in the soil (Yang, in preparation 1975).

Depth Below Surface (cm)	Greens (ppm)	Fairways (ppm)
0 - 4	647.9	83.4
4 - 8	238.9	50.1
8 - 12	102.2	30.3

*1 ton = 2000 lbs = 0.91 metric tons

The average application rate for golf courses is 294 pounds of lead arsenate per acre. It should be noted that the range of values for golf course soils was 6 to 3,330 ppm of lead.

Lead arsenate was used as an insecticide in the growing of tobaccos until about 1950. As a result of the changing patterns in pesticide usage, tobacco has decreased from an average lead level of 130 ppm in the 1950's to 20 ppm in the 1960's and appears to have leveled off at 20 ppm in the 1970's (Buckley, et al., 1973).

An example of the acute effects of high blood-lead concentrations in a pregnant mother and her newborn infant are well illustrated in a case reported by Palmisano, et al., (1969) in which these authors also state that lead has a "devastating effect on reproduction and pregnancy since it most commonly causes sterility or early spontaneous abortion." It has been clearly shown that lead crosses the human plancenta and may cause untoward effects in the fetus (Karlog and Moller, 1968). Around the beginning of this century it was recognized that women employed in the lead trades often produced liveborn infants who were small, weak, and neurologically damaged (Cantarow and Trumper, 1944; Angle and McIntire, 1964).

CRITICAL SOURCES

Lead, which is found in the earth's crust, also occurs naturally in the atmosphere and hydrosphere as a result of both physical and chemical processes. Man's activities, of course, introduced the greatest quantities of lead and lead compounds into the atmosphere and hydrosphere. For example, lead or its compounds can enter the environment at any stage during the mining, smelting, processing, and use of this metal and its derivatives. Additionally, lead is stable and incrementally accumulates as a waste or contaminant.

Increases in lead use have been on the order of 3% per year over the last decade, and the total annual consumption of lead in the United States, summarized in Table 11, is approximately 1.3 million short tons. However, most of this lead is not in a form or use likely to pose an environmental or health threat. In fact, over 40% of the annual consumption of lead comes from recycling.

Major sources of lead causing or likely to cause adverse environmental or health effects include exhaust from the use of leaded gasoline and other fuels as oil and coal; the use of paint, ink coloring, and other pigments containing lead; the use of lead in treating, processing, and packaging food; the use of lead in water pipes, ceramic pottery, batteries, and pesticides; the use of lead as a stabilizer in the manufacture of certain plastics; and the mining, smelting, processing, and terminal disposal of lead-containing materials. These and other sources bring large quantities of lead into man and his environment via air, water, and food.

The principal sources of atmospheric lead are listed in Table 12. Lead dusts entering the upper and lower respiratory tract are in the particle

size range of from less than 1 to greater than 10 micrometers. Although different particle size distributions and proportions emanate from these various sources, the resulting size profile, after gravitational and atmospheric interaction, is believed to be approximately a logarithmic normal distribution. Consequently, various sources of respirable airborne lead ultimately yield a somewhat similar size distribution profile (Lee, et al., 1968; Task Group on Lung Dynamics, 1966).

Table 11. LEAD CONSUMPTION IN THE UNITED STATES (Short Tons)*
(Information from National Academy of Sciences 1972)

Uses	1968 Totals
Metal Products	915,500
Pigments	109,734
Chemicals*	262,526
Coatings and Miscellaneous	41,030
GRAND TOTAL	1,328,790

*261,897 Tons are gasoline additives.

Table 12. EMISSION SOURCES OF LEAD IN AIR
(Information from U.S. Environmental Protection Agency Position on Health Implications of Airborne Lead, November 18, 1973. Environmental Lead and Public Health, Air Pollution Control Office, Publication No. AP-90, March 1971.)

Source	Estimated Emissions (Tons)* in 1968
Gasoline Combustion	181,000
Coal Combustion	920
Lead Alkyl Manufacturing	810
Primary and Secondary Smelting	811
Incineration of Solid Wastes	11,000
Other (pesticides, paint degradation, construction materials, inks, chemicals, etc.)	755
TOTAL	195,296

*1 ton = 2000 lbs = 0.91 metric tons

Patterns of lead emission to the atmosphere have changed in modern times. Emissions from coal burning and smelting have been checked by improved industrial controls, but these decreases have been more than offset by emissions from automobile exhaust fumes. Today, about 98% of the airborne lead that can be traced to its source comes from combustion of gasoline (National Academy of Sciences, 1972). It is estimated that 66% to 75% of the lead fuel additive is emitted into the atmosphere from motor vehicles, and over one-half of this emission becomes widely dispersed.

Automobiles are moving sources concentrated primarily in or near residential areas. If the most critical receptors presently known are assumed to be pregnant women and young children, and these receptors are also assumed to be distributed throughout residential areas, it appears logical that monitoring networks would be most efficacious in residential neighborhoods immediately leeward of the more heavily traveled streets and adjacent to major arterials. A correlation which might have general applicability can probably be established between traffic volume, distance from the traffic, and exposure levels in any urban area.

In addition, specialized monitoring stations are also desirable to cover major fixed sources of respirable lead pollution such as smelters, public incinerators, power plants, and agricultural areas using lead arsenate pesticides. Here, meteorological considerations would be important in properly locating monitoring stations in order to determine nearby residential exposure levels.

In considering all routes of lead to the critical receptor, concern lies in two areas: (1) total suspended particulates, and (2) dustfall containing lead, either of which can be ingested and respired due to human and climatic activity. The first category is in the size range under 10 micrometers, and the second category is in the range above 10 micrometers. Concern also must be given to particulate emissions from metallurgical processes, paint degradation, building demolition, and other processes and operations which contribute both to the general suspended particulate concentration and to more localized dust concentrations.

QUANTITATIVE MATHEMATICAL MODELS

A generalized model for a pollutant distribution which can be used for lead is presented below. In addition, submodels for various processes and compartments are needed for the special problems of lead, and several of these submodels have been developed for lead.

A qualitative representation of process in the pollutant-environment-receptor can be represented schematically in a pollution chain diagram. Some notations are included in the diagram which are explained below. This diagram and the mathematical model were developed for the United States Department of Health, Education, and Welfare by Lutz, et al., (1970).

Sources		Transport Pathways		Exposure Rate		Population Exposure
(S_{ik})	→	(P_{jk})	→	(D_i)	→	(E_{ijk})

The integrated exposure of the receptor from the stressor pollutant can be represented by the following equation:

$$E_{ijk} = \int_{t_1}^{t_2} S_{ik}(t)\, P_{jk}(t)\, D_i(t)\, dt$$

where E_{ijk} = the population exposure from pollutant i over time t_1 to t_2 through pathway j to man at location k

S_{ik} = the time varying source strength of the pollutant i at location k (It represents the amount of pollutant released into the environment that could ultimately become a stressor to the population.)

P_{jk} = the transport pathway term for pathway j at location k (It is a fraction, less than 1, representing the environmental dilution processes from release at the source until exposure of the population.)

D_i = the exposure rate for the pollutant i (It includes the exposure rate that man would be subjected to from all modes of exposure and portals of entry.

In the preliminary stage of the model, ordinary differential equations are utilized. Development of the model is an iterative process of exercising the model in its preliminary stages, determining what changes are necessary, and investigating mechanisms that better represent the system. Most of this involves more detailed submodels. This iteration process includes sensitivity analysis of the parameters of the model to determine how sensitive the output of the model is to variations in the parameters of the model.

In this study, the approach used to develop the model was to represent the total transport of the pollutant from the source to an individual as a first phase, with the subsequent distribution in several body organs of the individual due to the principal modes of entry into the body - inhalation and ingestion.

A submodel for lead in the body describes the distribution of lead in several body organs (lung, blood, bone, liver, and kidneys) due to intake of lead from inhalation and ingestion (Lutz, et al., 1970). While the model is not a very detailed representation of the biological processes, the predictions of lead in the body organs were within the accuracy desired. The calculated blood-lead levels using the model were within a factor of two of reported blood-lead levels. The quantitative predictions of blood-lead

levels due to intake of lead by ingestion or inhalation were shown to be valid within the accuracy of this preliminary modeling effort.

A submodel was also developed by Battelle (Lutz, et al., 1970) for young children having pica behavior and ingesting abnormally high amounts of lead as in paint chips. Since ingestion of lead via foodstuffs was stated to not vary significantly, no submodel for foodstuff ingestion was developed.

A model for lead distribution and movement through a watershed was developed in the University of Illinois, Research Applied to Natural Needs study on lead (Rolf, et al., 1972). This model divides the watershed into terrestrial and aquatic subsystems or zones and into additional subsystems based on degree of lead exposure. The model distributes the lead to nodes that are connected to the atmosphere, and the lead contained in each node is distributed as follows:

$$y_{ij}(t) = x_i(t) \frac{d_{ij}\, s_{ij}(t)}{\sum_{j=1}^{n} d_{ij}\, s_{ij}(t)}$$

where y_{ij} = the quantity of lead moving from node i to node j during time period t

x_i = the quantity of lead in node i

d_{ij} = the flow, relative to other branches leaving i, from i to j

n = the number of branches leaving a node

s_{ij} = a seasonal factor for branch ij at time t (The seasonal factor is a step function to simulate a nonlinear function such as plant or animal growth.)

Once the relative flows between nodes (dij's) have been estimated, this type of model is highly versatile. Each zone can be considered a separate unit and different systems can be modelled by proper arrangement of units and changing the internal configuration of nodes and branches. This model was used to estimate future lead levels in the watershed areas of study.

An atmospheric lead transport submodel was developed and tested by Battelle (Lutz, et al., 1970). This model was tested using data on airborne lead concentrations in Cincinnati, Ohio, with lead in gasoline as the major source. The atmospheric transport model did not provide a very detailed representation of the atmospheric diffusion of lead; however, the predictions of airborne lead concentrations using this model were within the accuracy required. For an adult city dweller inhaling lead at an average concentration of 1.0 $\mu g/m^3$ and ingesting 300 µg of lead daily via foodstuffs, the predicted blood-lead level, from calculations using the mathematical model, was 17.6 µg/100 g blood.

While the models and submodels were developed by the United States Department of Health, Education, and Welfare on a theoretical basis, they were developed to simulate real-world interactions closely to the extent of available data and knowledge. The predictions of the mathematical model are quantitative and are based upon a systematic utilization of basic data available as compared to guesses or intuition.

For the special problem of monitoring atmospheric lead from automotive exhaust, the described models do not provide sufficient detail to account for the variability from sources such as urban streets. This is required to develop a practical monitoring system, and this type of submodel was developed during the workshop. The submodel is based upon the work of Ott (1974) and is similar to the approach of Gilsinn (1972).

The problem of properly monitoring an atmospheric pollutant concentration can be better defined if a conceptual framework is constructed. The purpose of such a framework is to facilitate the design of an overall network which ultimately treats routes of exposure through all environmental media.

For purposes of this submodel the overall lead particulate concentration that remains suspended in air (non-setteable) in a particular urban area is assumed to be comprised of four different components:

(1) Contribution from mobile sources,

(2) Contribution from stationary sources,

(3) Contribution from other cities, and

(4) Geophysical background contribution.

The concentration measured at any location in the city and at any moment of time represents the sum of these four components. The first two contributions, the lead concentrations from mobile sources and from stationary sources, vary greatly with locations within the urban area while the last two, because they are very thoroughly mixed in the atmosphere, do not vary spatially across the city.

This approach, which is referred to as "component analysis," treats air quality at any location, $C_T(x,y)$, as being the sum of these four individual components:

$$C_T(x,y) = C_G + C_I + \sum_{i=1}^{n} C_{M_i}(x,y) + \sum_{j=1}^{n} C_{S_j}(x,y)$$

where C_T = total atmospheric lead concentration ($\mu g\ Pb/m^3$)

C_G = geophysical background concentration component ($\mu g\ Pb/m^3$)

C_I = long-distance extra-urban background concentration component ($\mu g\ Pb/m^3$)

$C_{M_i}(x,y)$ = component of lead concentration contributed by street i as a function of x and y distances from the street ($\mu g\ Pb/m^3$)

$C_{S_j}(x,y)$ = component of lead concentration contributed by stationary source j as a function of x and y distances across the city ($\mu g\ Pb/m^3$)

Figure 8 is a graphical representation of the equation. The vertical axis shows the atmospheric lead concentration ($\mu g\ Pb/m^3$), and the horizontal axis shows distance across the city (km). If effect, this figure provides a typical "cross section" of lead concentration as a function of horizontal distance X. The components of concentration given in the above equation are depicted on this figure. The total air-lead concentration C_T is the sum of these components.

Components C_G and C_I are treated as constants, since they do not vary with distance. Component $C_M(x,y)$ is assumed to have many maxima, one at each street. In Figure 8, each street may be viewed as perpendicular to the plane of the figure, with traffic flowing toward and away from the observer. Each of the narrow, tall maxima is the concentration "envelope" contributed by the street. The airborne lead concentration arising from vehicular exhaust is highest in the immediate vicinity of the traffic, dropping off rapidly with distance from the highway. This concentration component never quite reaches zero, however, because the contribution from the next street or highway soon begins to have influence. The height of each maximum is a function of traffic volume (vehicles/hour), road configuration, traffic acceleration, and micrometeorology. The heights of the maxima have been chosen to depict typical values. This picture represents, of course, only an "idealization." In actual situations, the symmetrical curves would be skewed somewhat due to the influence of wind. (For simplicity, this skewness has been omitted from the drawing.)

The last term in the equation represents the concentration component contributed by stationary sources of atmospheric lead. In general, stationary sources (smelters, power plants, incinerators) are more varied than mobile sources. Furthermore, some cities may have more than one lead smelter while other cities may have none. Due to this variability, "specialized" networks should be set up to monitor air-lead concentrations immediately around each stationary source. The distribution of these stations would depend on the size and the nature of the source as well as local meteorological characteristics. The purpose of a specialized network is to gather data on the highest concentration to which members of the general public surrounding this source might be exposed. Because more than one stationary source of atmospheric lead may be present in a given city, the last term in the equation is shown as a summation term. The concentration from any given stationary source is, of course, a function of the horizontal distance from this source and other factors.

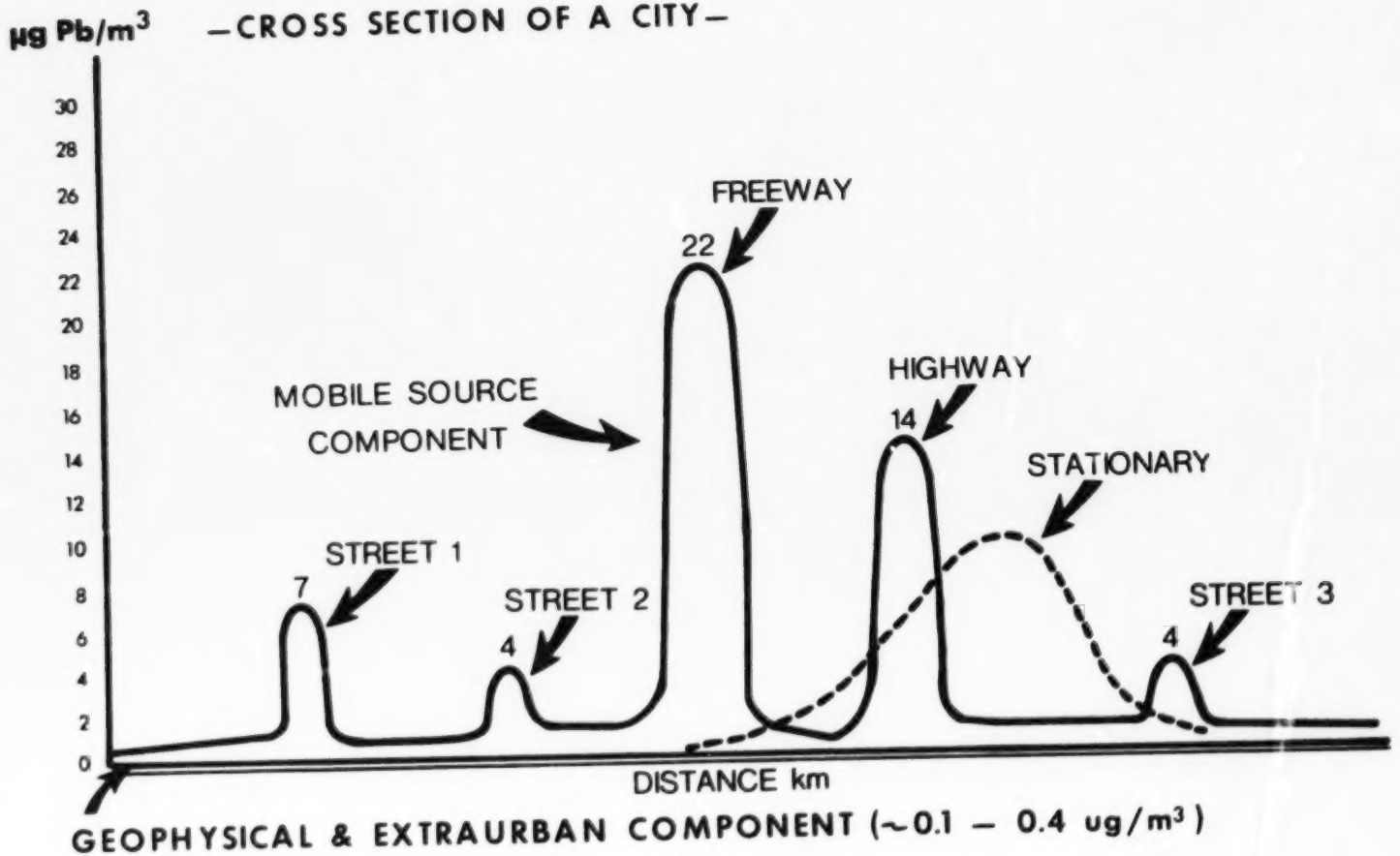

Figure 8. Spatial Distribution (Suspended Fraction) of Lead Concentration (Geometric Mean) in Air

If the purpose of the network is to monitor exposure of the general population to the highest concentrations, it would be desirable to locate monitoring sites where: (1) concentrations are highest, and (2) large numbers of people congregate for relatively long periods. Thus, we are interested in the joint occurrence of both high concentrations and large numbers of people for long periods. A conceptual way to treat this problem is to introduce the "population occupancy" function. This function is the product of the number of people at a given location in the city and the time they spend at this location. A hypothetical example of such a function is shown in Figure 9 (solid curve) along with the total concentration distribution, i.e., the sum of the components in Figure 8 (broken curve). Typical units are "people-hours," and these data can be obtained from an in-depth urban survey of population activity in the urban area. (Such a survey probably would include aerial photography.)

The joint occurrence of both high atmospheric concentrations and high population occupancy for long periods of time at a given location would be the basis for establishing a sampling station at that location. Locations where these two variables were jointly large would be ranked. High-volume samplers would then be installed, along with cascade impactors in some instances, at locations receiving the highest ranking.

It should be noted that this suggested modeling approach does not take into account the influence of resuspension of settled lead particles. At this time insufficient information prevents inclusion of this important contribution.

BASIC RESEARCH REQUIREMENTS

As a result of the review of the available literature and a consideration of information requirements for the design of an integrated monitoring system, many research requirements have been identified. Some of the more urgent research requirements for critical receptors and transport pathways are outlined in this section.

1. Research on Critical Receptors

   a. Conduct research on lead exposure to children playing, eating, and in other behavior with regard to lead intake from air, dust on hands, food, and other contaminating methods.

   b. Determine the contribution of lead from the mother to the newborn child. This should include pathological studies and blood-lead level determinations in stillborn and aborted fetuses, particularly for brain and nerve damage in those with high blood-lead levels. Umbilical cord blood-lead level determinations are needed.

   c. Information on the respiratory physiology and ventilatory absorption, retention, and excretion of lead in the infant and

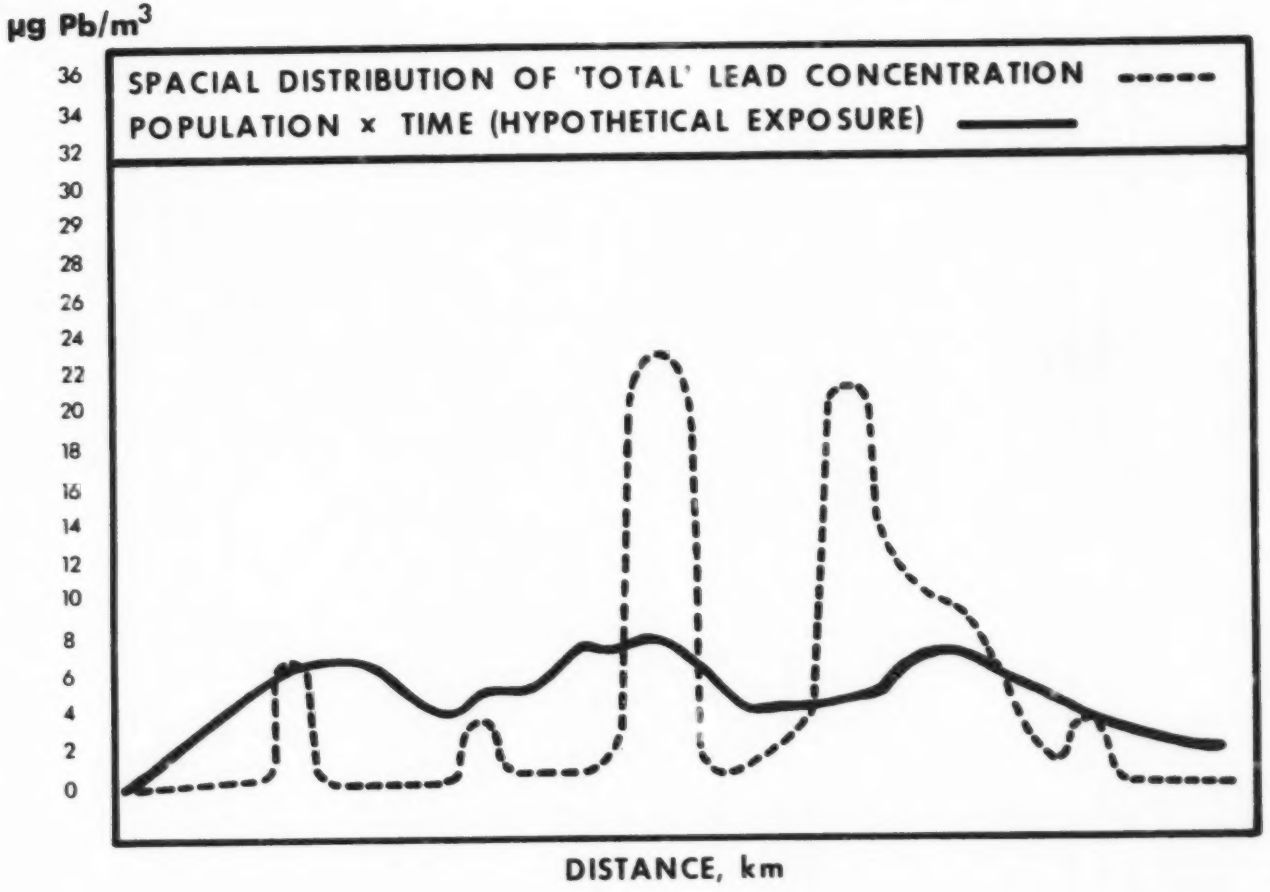

Figure 9. Hypothetical Exposure Cross Section to Total Atmospheric Lead in a Typical Urban Area

child is unavailable. Research to define minimum and maximum daily rates of inspired lead under resting and various activity-level states is required. No data exist on the relations between the particle size of airborne lead, ventilatory rates, and alveolar exchange, or the degree of respiratory absorption in children. Existing information is largely a poor estimate based upon model information regarding suburban and rural children and adults compared to urban children and adults.

d. Solubility factors (availability for absorption) should be elucidated for various pollutant compounds, such as lead, within the human body, particularly in children. For example, the solubility of dusts and paint chips in digestive juices may be significantly different with consequently different gastro-intestinal absorption rates.

e. The effects of recent changes in recreational activities need to be assessed; for instance, jogging and bicycling in areas of heavy traffic and swimming in polluted rivers and lakes. Additionally, research is needed to determine the most representative vertical sampling zone or profile for monitoring exposure levels.

2. Research on Transport Pathways

a. Definitive models need to be developed to relate lead concentration, traffic volume, and distance from streets and highways as well as to determine the vertical lead distribution profile and the effects of local meteorology.

b. Research is needed on the resuspension of particulates in the household. The extent to which settled particulates reenter the air in a household is poorly defined. It may depend on house cleaning, human traffic, and other activities. This may vary from household to household. Tracer studies could be undertaken to better define this phenomenon.

c. Multi-variate analyses are needed for heavy metals in air particulates and gaseous air pollutants. Multi-variate analyses would determine the correlations and relationships of these variables with each other for different cities and different site locations. Carbon monoxide and lead concentrations are correlated with traffic volume, and effects observed may be due to lead alone or lead combined with carbon monoxide (and perhaps with other heavy metals).

d. Determine how lead is added to processed foods and recommend measures to reduce this source of dietary lead intake.

RESEARCH MONITORING REQUIREMENTS

In addition to basic research, which develops concepts and finds underlying natural mechanisms, there is also a need to apply the basic research results to specific and highly individualistic situations. In order to do this, very specific information is required about each situation. Much of the information needed to implement monitoring system designs, both current and hypothetical, is unavailable. The following section outlines the various monitoring research requirements foreseeable for an integrated monitoring system applied to lead.

1. Research on Critical Receptor

    a. Research monitoring of blood-lead levels and other parameters in children is required in areas of high levels of lead in the air and in dustfall to determine more accurately their part in childhood plumbism.

    b. Research monitoring is required on blood-lead levels in expectant mothers and new mothers in areas with high exposure risk to lead. Correlations should be made between mothers' blood-lead levels and that of newborn infants.

2. Research on Transport Pathways

    a. Research monitoring should be conducted in critical target cities to monitor lead in air at 1 meter above the surface in inner city areas, near freeways, in heavy traffic, near gasoline stations and bus stops, and inside of highly exposed houses.

    b. Lead in dustfall should be measured in the same areas as above.

    c. Research monitoring is required to test and verify models which would relate lead concentration, traffic volume, and distance from streets and highways. There is evidence that the highest atmospheric lead concentrations occur very close to streets and highways, decreasing rapidly as distance from the highway increases. Therefore, mathematical relationships (with narrow confidence intervals) must be developed which would ultimately enable the concentration very near streets to be estimated for highways in any given city when just the traffic volumes are known.

    d. Vertical profiles should be determined for airborne lead concentrations. Monitoring instruments often are located at very different heights. Therefore, it is necessary to fully document the change in lead concentrations with elevation. A detailed microscale monitoring research investigation is recommended for the purpose.

e. A relationship should be established between outdoor airborne lead and indoor airborne lead. Most members of the general public, including critical receptors, spend most of their time indoors. Thus, more accurate models are needed which relate outside lead concentrations to inside lead concentrations. These can be established by an intensive indoor/outdoor monitoring research study using houses in different locations, both near and far from streets.

f. Research monitoring should be conducted around stationary sources of lead pollution such as power plants (especially those burning coal which has a high heavy-metal content), incinerators, factories, or smelters when these are near high population or residential areas.

g. Air-lead concentrations and blood-lead concentrations of drivers in traffic should be measured. Drivers and passengers in automobiles may receive high air-lead exposures; thus, comprehensive data are needed on lead concentrations inside vehicles moving in traffic. (These vehicles should include school busses). Estimates should be made of the integrated average concentrations for persons spending 2 to 8 hours in traffic.

h. Information documenting values of absorbable lead within foods consumed in the diets of Americans, including all subgroups and subcultural dietary considerations, is needed. Population groups (especially children) likely to consume large quantities of potentially high-lead content foods and beverages should be determined.

   Diets disproportionately composed of foods that are particularly prone to contain or carry lead (e.g., legumes, fruits, shellfish) should be monitored to establish base lines and/or trends.

i. Many older sections of cities have lead water pipes. These should be identified and the water monitored to obtain exposure data for this source.

DESIGN OF AN INTEGRATED MONITORING SYSTEM

All people are exposed to environmental lead, and practical considerations must prevail in establishing a monitoring system. It is, therefore, necessary to rank the probable receptors according to environmental, physiological, and behavioral criteria. In the case of lead, clinical evidence overwhelmingly indicates the young urban child as the most critical receptor. This may be due primarily to the combination of behavioral patterns and the increased availability of environmental lead. Additionally, some recent evidence also indicates that the child may absorb 5 to 10 times more lead and

suffer adverse effects from lead at lower blood concentrations than adults do.

Considerable scientific data exist about lead in numerous disciplinary fields. However, a marked deficiency of respective intercommunication between disciplines and the subsequent interpretation and analysis of these data remains at the present time.

It is strongly recommended that an interdisciplinary team approach be utilized as a part of any new monitoring system design. Computer systems should be used. Computer systems should be envisioned and programmed prior to the onset of monitoring for correlative and objective analysis of all information to be gathered. Such an information system should permit future modification, growth, and reorientation for continued use and cost benefit.

When the design model is complete, feasibility testing is recommended for this monitoring system in several representative United States communities as soon as practicable. Such an opportunity to correlate interdisciplinary efforts can be utilized to obtain the needed dependent extrapolation from studies performed on adults.

It is technically feasible to design an integrated monitoring system for various pollutants after study of the variables and assignment of tentative priorities. An integrated monitoring system designed on the basis of modifications to a lead model system would probably have general applicability because lead is universally prevalent in air, water, and food cycles.

IV. CONCLUSIONS

As a result of an analysis and review of the concept of an integrated monitoring system for pollutants, using the test case of lead, the following conclusions are presented:

1. The concept of identifying critical receptors, transport pathways, and sources appears to be a valuable method for developing more efficient monitoring.

2. Design of a monitoring system which will provide usable information directly for control of critical sources is very valuable.

3. The concept provides a framework for logically organizing existing information and provides a means of analysis to determine missing information.

4. Appropriate models appear to be very useful for quantifying the relative importance of various factors, especially major transport pathways. More work is needed in evaluating and actually using and testing models.

5. The concept and models provide the basis for designing basic research and research monitoring programs to provide needed quantitative data.

6. Lead was a suitable pollutant to use as a test case, and the criteria used for selecting it for this purpose appear valid. Many gaps were discovered in the data on lead.

7. It is recommended that the evaluation of the integrated monitoring concept be continued as follows:

a. Review mathematical models and use the data for lead assembled in this workshop in the model and submodels selected. Test the models, and determine the quantitative data still needed for lead.

b. Use another test-case pollutant for further evaluating the concept of the integrated monitoring system. Another test case could be the cholinesterase monitoring system for organophosphates used by the Department of Defense.

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